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HEALTH EFFECTS OF ACID RAIN PRECURSORS

HEARING

BEFORE THE

SUBCOMMITTEE ON
ENVIRONMENTAL PROTECTION

OF THE

COMMITTEE ON
ENVIRONMENT AND PUBLIC WORKS
UNITED STATES SENATE

ONE HUNDREDTH CONGRESS

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HEALTH EFFECTS OF PRECURSORS OF ACID RAIN

TUESDAY, FEBRUARY 3, 1987

U.S. SENATE,
COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS,
SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION,
Washington, DC.

The subcommittee met, pursuant to notice, at 9:30 a.m., in room SD-406, Dirksen Senate Office Building, Hon. George Mitchell (chairman of the subcommittee) presiding.

Present: Senators Mitchell, Baucus, Graham, Chafee, Stafford, Durenberger, and Pressler.

OPENING STATEMENT OF HON. GEORGE J. MITCHELL, U.S. SENATOR FROM THE STATE OF MAINE

Senator MITCHELL. The hearing will come to order.

In the last few years there has been a great deal of research into adverse health effects of acid rain precursors such as sulfur dioxide and nitrogen oxides. Today's hearing focuses on these effects.

Data suggest that sensitive populations, such as asthmatics, experience increased ill-health due to existing, ambient levels of sulfur dioxide.

A Harvard University study demonstrates that as levels of air pollution increase, so do hospital admissions of children. In areas like the Northeast, where there is cold weather and higher ambient levels of sulfur dioxide, sensitive adults and children are more vulnerable to wintertime illnesses.

These and other data demonstrate that the health risks associated with exposure to air pollutants may be greater than previously believed.

At a hearing last year, there was testimony that exposure to sulfur dioxide and nitrogen oxides produce the same effects as cigarette smoking. We have a national campaign against cigarette smoking, and spend large sums of money to discourage people from smoking, yet we do nothing to control these acid rain precursors. The effects are the same, but the Administration's commitment is not.

The health data, when combined with actual damage to our waters and potential damage to our forests, suggest that legislation is needed. The fact that emissions of these pollutants are increasing suggests that legislation is needed now.

When I introduced acid rain legislation six years ago, I was told by the Administration that more research was needed before congressional action was warranted. We have had research and the ironic result is that the more research we do, the more the case of acid rain legislation becomes clear, yet the Administration's position is that we need more research. In the December issue of the EPA Journal, Lee Thomas, the Administrator of the EPA, commenting on the greenhouse effect and ozone depletion, said and I quote:

> If we wait until health and environmental impacts are manifest, it might be too late to take adequate steps to address these problems. We must realize that there will always be scientific uncertainty associated with these complex problems. We will have to be prepared to act despite these uncertainties.

If that is an adequate standard for addressing this air pollution problem, surely it is the standard for addressing acid rain. I, for one, am prepared to act this year to pass acid rain legislation, and invite the other members of the committee and the Administration to join me.

I would like to place in the record at this point statements from Senators Moynihan and Lautenberg.

[The statements referred to follow:]

OPENING STATEMENT OF HON. DANIEL PATRICK MOYNIHAN, U.S. SENATOR FROM THE STATE OF NEW YORK

Mr. Chairman, I think it most appropriate that the Environmental Protection Subcommittee begin at this early date in the 100th Congress to consider the effects on health of acid rain and its precursors. I look forward to hearing from the eminent scientists who will testify before us today; we have much to learn.

Certainly the notion that sulfurous emissions are a hazard to health is not new. Edward I banned the burning of coal in London in the 14th Century. Circumstances in the years since then have dictated that coal and other fossil fuels would be burned in great quantity—despite Edward's views. We have learned in due course that both sulfates and oxides of nitrogen are produced in the process, noxious agents with potent effects on human health.

I am concerned in no small degree with the health effects of acid rain and its precursors. In part, that is why I authored the Acid Precipitation Act of 1980, the law that set much of our acid rain research in motion. The only acid rain law we have now got.

But, Mr. Chairman, my understanding is that the witnesses before us today will be speaking of human health alone. What of the health of Nations?

It has been abundantly clear that the Canadian people and the Canadian government have deeply held views on the necessity of prompt and comprehensive acid rain controls. Passions run deeper on little else. And yet the Reagan Administration has dealt with the Canadians most shamefully on this, an issue they hold most dear.

Not only has the health of our relationship with our most important trading partner been damaged; the health of the Canadian government itself has been put in mortal danger on account of it. Mr. Mulroney reached out to his friend, the President of the United States. Their envoys, Drew Lewis and Bill Davis, produced a joint report recommending a $5 billion acid rain control technology development and demonstration program. Both leaders endorsed that report. And the result for Canada's Prime Minister: a slap in the face, and a Government very much at risk.

Mr. Chairman, the program that Mr. Lewis put forward is not enough. But it represents the first time that the Reagan Administration has gone on record to say that acid rain is a problem deserving of a solution, even an expensive one. More importantly, it represents the commitment of the United States Government. A commitment that this Administration appears prepared to honor in the breach.

Mr. Mulroney put his government on the line when he accepted our promise. We put his government in jeopardy by failing to follow through. We committed to $2.5 billion from U.S. industry and a like amount from the U.S. Treasury. But after not much time, our Energy Secretary was heard to say that we could not afford a new $5 billion program. And now it appears that the Administration is primarily relying on the worthwhile—but preexisting—Clean Coal technology demonstration program within the Department of Energy.

Nothing can be more important than human health, but the health of Nations and the relationships between them is vital to our welfare as well. Odd that the Department of Energy can imagine $6 billion for a "Superconducting Super Collider," an important program for basic physics research, but cannot imagine $5 billion for a program to bring acid rain under control and, perhaps, save a government and our relationship with it.

I submit that this too is a health effect of acid rain, an important one deserving our attentions.

OPENING STATEMENT OF HON. FRANK R. LAUTENBERG, U.S. SENATOR FROM THE STATE OF NEW JERSEY

Mr. Chairman, I commend you for holding this hearing today about the health effects of acid rain. I was pleased to join you in introducing legislation (S. 321) to control this problem. I look forward to working with you and others on the committee to address this pervasive threat to the Nation's health and environment.

100,000 tons of sulfur dioxide pollution is spewed out over our cities, forests, rivers, and lakes every day. Left unchecked, these emissions will increase in the years ahead.

Acidified lakes in the Adirondacks began to draw attention in the late 1970's. In 1984, the Office of Technology Assessment reported that 9,000 lakes and 117,000 miles of streams are at risk in States east of or bordering the Mississippi. And scientists are now discovering that the problem extends west to Colorado, California, and the State of Washington. Evidence suggests that this forest damage is linked to acid rain related pollutants.

New Jersey also has seen the effects of acid rain and its related pollutants. Decreased growth and foliage changes in the Pine Barrens may be attributable to pollutants. Correlation of acidified streams and reduced productivity of pines in the Lebanon State Forest was the first indication on the eastern seaboard that acid rain may be damaging our forests. Also, in New Jersey's Kittatinny Mountains three lakes are threatened. Acidity levels endanger trout and bass breeding.

Mr. Chairman, the evidence is building on the health effects of air pollutants related to acid rain. Evidence suggests that chronic coughing is more common in regions with higher sulfur dioxide and particulate pollution. Also, serious respiratory problems result from increased ozone concentrations.

The ozone problem is of particular significance in New Jersey, which unlikely to meet the 1987 Federal deadline for ozone reduction. New Jersey has tough State laws. But New Jersey's ozone problem is not all New Jersey's making. Prevailing westerly winds carry the components of ozone—nitrogen oxides, hydrocarbons, and other chemicals—from Midwestern States. They blow in from States with weaker environmental standards for utilities and industrial facilities.

Enough evidence is in. It is time for Congress to act. I look forward to working with you, Mr. Chairman, and the committee, in the battle to protect our forests, lakes, and health from these insidious forms of pollution. I welcome the witnesses here today, and look forward to hearing their views on this critical issue.

Senator MITCHELL. With that, we will proceed to the panel. The listed panelists are: Dr. Richard Narkewicz, Dr. Thomas Godar, Dr. Anthony Robbins, and Dr. Phillip Landrigan.

Gentlemen, good morning, and thank you very much for coming. We appreciate your willingness to provide your insights to this committee.

I would ask that you limit your opening oral remarks to five minutes. Your entire written statement, if you have one, will be inserted in the record for review by the Members of the committee. This box has a green and a red light, and the red light will come on when five minutes are up. I would appreciate it, when you see the red light come on, if you would bring your remarks to a conclusion, consistent with the points that you want to make. There will be a question and answer period, and ample time for you to make all of the points that you feel are important.

With that, we will begin with Dr. Narkewicz. Welcome, Doctor.

STATEMENT OF DR. RICHARD M. NARKEWICZ, M.D., AMERICAN ACADEMY OF PEDIATRICS

Dr. NARKEWICZ. Thank you, Mr. Chairman, and committee Members.

I am Dr. Dick Narkewicz, President-Elect of the American Academy of Pediatrics, and a practicing pediatrician from Burlington, Vermont. It gives me great pleasure to speak with you today on behalf of the Academy, which represents over 30,000 pediatricians committed to improving the health and well-being of infants, children, adolescents and young adults.

As a practitioner for over 20 years, the message is simple and to the point. It is hazardous to the health of children to breathe polluted air. Air pollution is a complex issue because the health effects depend upon many ingredients—the specific pollutant, the intensity of the dose and duration of the dose, and the specific characteristics of the patient involved.

The specific air pollutants of concern in this testimony, Mr. Chairman, are the oxidizing agents such as ozone and nitrous dioxide, and reducing agents such as sulfur dioxide. These are some of the ingredients of acid rain.

While the ecological, environmental effects of acid raid are much discussed, the focus of this testimony is that these pollutants contribute greatly to the development of human lung disease. These pollutants cause disease in children, and significantly aggravate underlying respiratory conditions.

It is of great significance when you consider that pediatric pulmonary disease accounted for 21 percent of all hospitalizations in children under 15, and 29 percent of total hospital days in 1983. Combined figures from 1981 and 1982 indicate 4.5 million office visits. It is a staggering cost in morbidity and dollars.

The diseases we are talking about are asthma and bronchitis. These diseases can be caused or aggravated by many factors, especially air pollution. Children are particularly vulnerable to these pollutants because of several reasons.

Number one, children's airways are much smaller than those of adults. The diameter of an adult bronchial tube is eight times larger than a child's bronchial tube. Therefore, you can easily see that breathing a pollutant that causes minor swelling and diminished diameter in an adult bronchial tube, may produce dangerous obstruction in a child or infant.

Infants and children breathe more rapidly and, therefore, move more pollutant throughout the lungs. The repair process of children for damaged air ways is less efficient than that of adults, and the immunologic immaturity of children contributes to more respiratory disease. The obvious risk is that children's exposure to pollution will be longer, because of their life span, which potentially exposes them to more long-term crippling events.

Mr. Chairman, I see many of these children with asthma and wheezy bronchitis in my practice. I consider air pollution in any child suffering from respiratory difficulty. Nowhere in medicine does the axiom "an ounce of prevention is worth a pound of cure" play a more dramatic role than in pediatric pulmonary disease.

I spend the major part of my time teaching the child and the parents to avoid the aggravating causes of asthma and bronchitis because once the wheezing and gasping has started, it is difficult to control, and usually leads to one or many office visits, often hospitalization, and sometimes death.

There is clear evidence that the underlying hyper-reactivity of the small bronchial tubes of asthmatics, and those with bronchitis, are aggravated by pollutants such as the oxides of nitrogen, sulfates, and ozone.

In our role as practicing pediatricians, we are frustrated because we can't completely protect our patients from these irritants. Thus, we are forced to give our patients only symptomatic advice, which is the following: Stay indoors at times of severe pollution; keep the room as cool as possible; use recirculated air if possible; no smoking in the home; and drink plenty of fluids.

Pediatricians have a duty to educate the public about hazards to the health of children. I would like to reemphasize to this committee that the ingredients of acid rain have been shown to have considerable adverse effects on the respiratory track, especially in children. They contribute greatly to morbidity and quality of health for children.

The sources of the pollution are being identified, and the technology is developing to eliminate the pollutants. Doesn't it make more sense to eliminate the source of a health problem, if possible, rather than having children suffer and then attempting to treat the resulting symptoms?

The Academy is grateful for this opportunity to work with your committee in efforts to identify, reduce, or eliminate the major sources of acid air pollution. We recommend that strict measures to control emissions of sulfates, ozone, and the oxides of nitrogen from both industrial and automotive sources be implemented and enforced.

If this goal is achieved, the health benefits will be especially enjoyed by the children of this nation who depend upon you for their public representation. Thank you, Mr. Chairman.

Senator MITCHELL. Thank you very much, Dr. Narkewicz.

Dr. Godar.

STATEMENT OF DR. THOMAS GODAR, M.D., PRESIDENT-ELECT, AMERICAN LUNG ASSOCIATION

Dr. GODAR. Thank you very much, Mr. Chairman.

Mr. Chairman, and Members of the committee, I am Dr. Thomas Godar, Director of the Pulmonary Disease Section at St. Francis Hospital and Medical Center in Hartford, Connecticut. I am Associate Professor of Medicine at both the University of Connecticut School of Medicine and Yale University School of Medicine. I am an active, practicing pulmonary physician.

I am presenting comments today on behalf of the American Lung Association. In 1980, the Board of Directors of the American Lung Association developed its policy on the reauthorization of the Clean Air Act. Our principal concern then, as now, was the strengthening of the provisions of the Act to better control air pollution and protect public health.

I, and my colleagues in the American Lung Association and its medical section, the American Thoracic Society, have testified on numerous occasions before this committee on the subject of air pollution effects.

We have provided the committee with information demonstrating clearly the contribution of air pollution to the development or exacerbation of various lung diseases. The American Lung Association continues to place the highest priority on preserving and strengthening the Federal Government's program to control air pollution.

I appear before you today again with the very strong message that air pollution can and does have adverse impact on health. In some cases, this impact affects large populations. In 1981, the ALA encouraged the committee to consider the need to strengthen the health protection measures of the Act. Today, my message is the same but with greater urgency. The striking advances in information concerning health effects in the last several years justify this urgency, as noted in your introductory remarks.

I would like to summarize these advances and strongly encourage the committee to hold additional hearings to review the extensive record of research findings that in my opinion should be the driving force for rapid reauthorization of the Clean Air Act. The research findings cited below are all clinical studies done at levels of air pollution commonly found in many regions of the United States today.

Witness the finding that healthy asthmatic subjects exposed to sulfur dioxide levels as low as 0.25 parts per million for exposure periods as brief as five minutes, when performing mild to moderate exercises, can develop symptomatic and objective evidence of bronchial constriction, that is a tightening of the air ways.

Witness the finding that exposure to levels of sulfur dioxide found in the ambient air combined with exposure to cold dry air potentiate the onset of symptoms in as little as two minutes. These effects are of particular concern in areas such as the Ohio River Valley where such levels of sulfur dioxide and severe cold weather are common in the winter months.

Witness the finding that short-term exposures to 0.3 parts per million of nitrogen dioxide potentiates exercise-induced bronchial spasm and airways hyper-reactivity in healthy asthmatic subjects. Cold air is also found to potentiate this effect.

Witness the finding that, when combined with intermittent heavy exercise, exposure to ozone levels as low as 0.12 parts per million can cause symptoms of airways irritation and decreased lung function in healthy subjects. These effects are reproducible in the same subjects when re-exposed several weeks to one year later. In other words, there is reproducibility, there is no evidence of adaptation.

Witness the finding that exposure to 0.12 parts per million ozone in healthy subjects for a period typical of the normal workday, not yours or mine, or our good pediatrician colleagues, but a seven-hour day, for persons employed in outdoor jobs causes very significant decrements in lung function averaging reduction in forced expiratory volume in one second of half a liter. If your forced expiratory volume were four or five liters per second normally, this represents a 10 to 15 percent reduction in actual measurable lung function.

Legislative proposals providing for reductions in total emissions of sulfur compounds as well as emissions of other pollutants are likely to have substantial public health benefits. Reduction in source emission rates will reduce the magnitude and frequency of short-term sulfur dioxide exposures. Also, by reducing sulfur dioxide emissions, concentrations of sulfate particulate matter will be reduced, resulting in fewer morbidity effects.

However, to sufficiently protect health, including the sensitive population of asthmatics, current findings document the need to limit exposures to less than 0.4 parts per million of sulfur dioxide for durations of no more than five to ten minutes. Translation of this into a short-term, one-hour standard, including provision for an adequate margin of safety, would require a standard no higher than 0.20 parts per million.

To provide definitive health protection, the National Ambient Air Quality Standard for sulfur dioxide must be strengthened by the addition of a short-term standard to insure protection against adverse health effects at all locations in the United States. The Ambient Air Quality Standard provides the primary mechanism for guaranteeing health protection. ALA firmly believes that the NAAQS program is an essential component of any program designed to protect public health, including sensitive populations.

I would like to conclude my comments by indicating that in previous testimony we have documented that 20 percent of the population are sensitive to air pollutants, and there are health effects, if I could briefly list them. They are the pediatric population less than two years of age; pregnant women; patients with asthma, chronic bronchitis, and emphysema; patients with a history of myocardial infarction, angina pectoris, and other forms of coronary artery disease; and persons 65 years of age and older.

I believe that what is required is a comprehensive reauthorization of the Clean Air Act, and the ALA will offer its scientific and medical expertise in any way possible to assist you in assessing the complex health issues and making appropriate changes in the Act for resolution of these problems.

Thank you for this opportunity.

Senator Mitchell. Thank you, Doctor.

Before calling on Dr. Robbins, we are pleased to be joined at this hearing by Senator Baucus, who is the Chairman of the Toxic Substances Subcommittee of the full committee.

Senator Baucus, do you have an opening statement?

OPENING STATEMENT OF HON. MAX BAUCUS, U.S. SENATOR FROM THE STATE OF MONTANA

Senator Baucus. I do, Mr. Chairman, I will ask that it be included in the record, and to say that I am very happy that these hearings on health effects of acid rain are being held.

We all know the environmental problems that acid rain causes, but by large we, in this country, have lost sight of the potential adverse health effects. I think that it is a very appropriate hearing, and I look forward to the testimony of the witnesses.

Thank you, Mr. Chairman.

[Senator Baucus' statement follows:]

OPENING STATEMENT OF HON. MAX BAUCUS, U.S. SENATOR FROM THE STATE OF MONTANA

Mr. Chairman, today's hearing begins in earnest a process that hopefully will lead to the reauthorization of the Clean Air Act.

The promise of the Clean Air Act is to protect public health.

In 1970 when the Clean Air Act was enacted, a process was put in place to set standards to protect public health. All parts of the country were then to progress towards attaining that standard.

It is troubling that ten years after the last major reauthorization of the law, we may be losing sight of the goal to protect public health.

EPA, for example, has had the sulfur dioxide standard under review for almost four years. Yet no decision is forthcoming.

There is growing concern that the current ozone or "smog" standard does not protect public health.

Somewhere between 45 and 100 million Americans are breathing dirty air. It is clear that there are more children with decreased lung capacity and weakened health.

The Clean Air Act envisions a process where, as our understanding of the health effects of various air polutants grew, the Environmental Protection Agency would change the standards. This review was to occur every three years.

It hasn't happened. The Environmental Protection Agency has lost sight of its mission. While the tone of the Agency has changed since the early years of this administration, its action to protect air has not.

Acid rain has long been a concern of this committee. The American public wants clean air. Seventy-nine percent of the American public believe acid rain is a serious problem.

Whenever, I can, I hike Montana's wilderness areas. Nothing refreshes me more than standing on top of a mountain. The air is so clear a person feels like he can see forever.

The feeling is important. But the real joy of a mountaintop in Montana is knowing that you are breathing clean, pure air free of air pollutants. All Americans should enjoy clean air with every breath.

There is no real distinction between protecting public health and protecting the environment.

It is fitting that today's hearing focuses on health as the proper place to begin the debate.

Senator MITCHELL. Thank you, Senator Baucus.

We are also joined by Senator Chafee, who was the chairman of this subcommittee for six years and is responsible for most of the major environmental legislation that has been enacted in this country in the past several years. He is now the Ranking Member of the subcommitteee

Senator Chafee.

OPENING STATEMENT OF HON. JOHN H. CHAFEE, U.S. SENATOR FROM THE STATE OF RHODE ISLAND

Senator CHAFEE. Mr. Chairman, thank you very much.

First, I want to thank you and commend you for holding these hearings. It is not a surprise based on the experience and the deep interest you have shown on this subcommittee for so many years. Under your leadership, I am absolutely certain that we are going to move ahead with the whole acid rain problem.

I particularly want to commend you for these hearings today, concentrating on the health aspects of it, because, as Senator Baucus mentioned, we have had a good deal of testimony dealing with the environmental effects; the pollution on lakes and streams, and the effect; on our soil, but the particular area of health has been dealt with, in the past, in connection with the Clean Air Act in general, now we are addressing it specifically.

Mr. Chairman, I think that it is splendid that you are doing this. It is further indication of the deep concern you have had in all of these areas for so many years. I want to thank you and commend you for it.

Senator **MITCHELL**. Thank you.

Dr. Robbins, welcome.

STATEMENT OF DR. ANTHONY ROBBINS, M.D., PAST PRESIDENT, AMERICAN PUBLIC HEALTH ASSOCIATION

Dr. **ROBBINS**. Thank you, Mr. Chairman, and members of the committee.

I am Dr. Anthony Robbins. I am a Past President of the American Public Health Association, and I am here today in place of my colleague, and the President-Elect of the Association, Dr. Bailus Walker. I am also a former State Health Commissioner for the State of Vermont, a Health Director for the State of Colorado, and former Director of the National Institute for Occupational Safety and Health.

The American Public Health Association is an association of approximately 55,000 public health professionals. We are concerned primarily about the health effects of acid rain.

As the nation started to realize the consequences of acid rain, the shock set in from seeing dead lakes and dying forests, I think we tended to neglect the human health effects of the very same pollution.

These oxides of sulfur and nitrogen oxide, in complex interaction with other pollutants and with the environment, are now understood to produce acid rain. As the Congress tries to control these pollutants, and thus reduce acid rain damage, it is important that legislators be aware of the health effects of the very same pollutants.

There are approximately 25 million tons of sulfur dioxide emitted in the United States annually. Coal burning power plants, industrial boilers, smelters, and refineries are the major sources of sulfur dioxide. Control technologies may affect the dispersal of the emission, but may not affect the total quantity. High stacks have encouraged the use of high sulfur coal, and the effects are now distant, rather than local, where the coal is burned.

It is important, as you look at the health effects, to distinguish between two kinds, local or direct health effects, often referred to as pre-deposition effects from the sulfur dioxide and the resulting sulfuric acid. These compounds are often measured as fine particulates. I remember, a town in Colorado, in Salida, where a fertilizer plant was putting out enough sulfuric acid so that there were holes like that in every tin roof in the town.

Senator **MITCHELL**. Just so the record will be clear, because "like that" won't show up in the record. You mean the size of a quarter or a half a dollar?

Dr. **ROBBINS**. I am sorry, at least the size of a quarter, thank you.

Obviously, the best understood of these health effects, these direct and local health effects are on the lungs, and the two previous witnesses have dealt with that in some detail. The distant, or indirect, or post-deposition health effects, come from the same sulfur dioxide, now oxidized as sulfuric acid, and deposited, along with nitrous acid, as acid rain. These effects are caused by exposure to surface waters and shallow groundwaters that have become acidified.

Dr. Walker and my colleagues have reviewed the direct health effects—increased asthma attacks, increased acute respiratory problems, measurable decrements in pulmonary function, and particular problems for sensitive populations, young children, the elderly, those with respiratory disease, and those who have major exertion during work or exercise.

The indirect effects are less well understood, but are becoming evident. Obviously, the acid rain has affected the trees, has killed many lakes, but there are other effects as people consume the water from these sources because many of these lakes also serve as reservoirs.

Not all lakes have been acidified, not all are susceptible to acidification. In the State of Massachusetts, it is estimated that 1,000 of the 2,600 lakes are sensitive to the kind of acidification that will come from acid rain. Over 5 percent of them are now acidified. Maybe the most important one, Quabbin Reservoir, which provides drinking water to the City of Boston, is on the verge of being acidified.

What happens when the water is acidified?

Acid water has the property of being able to leach certain metals from the soil, from the silt, and from the pipes through which it flows. We get lead, copper and mercury, which may also be in the form of methyl-mercury and is more dangerous in terms of the health hazard put into solution. People have reported elevated aluminum levels as well.

We have a situation where these problems will continue and get worse as the presence of these acidified bodies of water leach more and more of these materials from the soil. At the present time, many public water systems simply add lye to the system and feel, at least, that they are protecting their old lead pipes, if they aren't changing the nature of these bodies of water.

What we also observe is that there has been more sensitivity in the health community, maybe because the remedy was more easily at hand, to doing something about the toxic materials in water than in the air.

Let me give you one brief example: we would close a water system if the public were getting the same levels of trichlorethylene from the water that the public in many cities routinely receives from the air at the present time.

If you have one part per billion of triychlorethylene in the air, it is equal to five micrograms per cubic meter. If someone, a normal individual, breathes 20 cubic meters per day and absorbs 50 percent of the trichlorethylene in that air, or 50 micrograms per day, that would be comparable to consuming two liters of drinking water, which is the normal rate of consumption, with 25 micrograms per liter, 100 percent absorbed, or 25 parts per billion in that water.

Right now, the EPA advice on TCE in drinking water is that the maximum contaminant level should not exceed five parts per billion. So we have, from air exposures, situations where we are getting amounts that would not be tolerated in drinking water.

I think, as we learn more about the local and direct effects of the air pollution that is producing acid rain at a distance, we will become increasingly concerned about the effects.

Let me conclude. With the pollution that causes acid rain, we have a situation where the local, direct health effects through the air, and the distant, indirect health effects through the drinking water, it is not sufficient simply to add lye to the drinking water to protect the old lead pipes. We must go to the source of this pollution.

I think the Congress faces a very difficult problem. The American Public Health Association believes there is sufficient evidence, but you must convince yourselves that more than trees are dying from the pollution that causes acid rain. At stake are people's health, and people's lives.

I would ask that the testimony prepared by Commissioner Walker be made a permanent part of the record.

Senator MITCHELL. That will be done, Dr. Robbins. Thank you.

Thank you, Dr. Robbins.

Dr. Landrigan.

STATEMENT OF DR. PHILIP J. LANDRIGAN, M.D., DIRECTOR, DIVISION OF ENVIRONMENTAL AND OCCUPATIONAL MEDICINE, MOUNT SINAI SCHOOL OF MEDICINE

Dr. LANDRIGAN. Thank you, Mr. Chairman.

My name is Dr. Philip Landrigan. I am Professor of Environmental Medicine and also Professor of Pediatrics at Mount Sinai School of Medicine in New York. I am Chairman of the Committee on Environmental Hazards of the American Academy of Pediatrics. Two years ago, I was Chairman of a Committee on the Epidemiology of Air Pollution, which was convened by the National Academy of Sciences and which produced the book, "Epidemiology and Air Pollution," which was published by the National Academy Press in 1985, and which I would commend to you.

I would like, this morning, to discuss with you the effects of acid air pollution on the health of children.

First of all, a word about current levels of pollutants. Levels of pollutants today are certainly lower than they were 20 years ago. In many respects, the Clean Air Act has been very successful. The so-called "killer fogs," such as that which occurred in Donora, Pennsylvania, are not seen any more in this country largely because of the good effects of the Clean Air Act.

On the other hand, the picture today is not uniformly benign. High local concentrations of air pollutants are seen still in relation to certain industrial complexes, and also along busy roadways. Sulfate concentrations are lower than they were in the past, but they are not negligible. In some parts of the country, particularly during the summer months, they rise to unacceptably high levels.

Perhaps most important, in terms of this morning's discussion, concentrations of ozone and of the oxides of nitrogen are not going down. In fact, there have been scattered increases in their concentrations over the past three to four years.

Now with regard to the health effects of acid air pollution, there are several converging lines of evidence, all of which suggest that acid air pollution has adverse effects on the lungs of vulnerable people within our population, particularly on the lungs of children. I would like to take a few minutes to review some of this evidence with you.

One of the major studies on the health effects of air pollution is the so-called "Harvard Six-City Study," which you referred to in your opening remarks. This is a study that researchers from the Harvard School of Public Health have been conducting over the past five years or so, following populations in six cities across the country, which span the range in air contamination from very clean to quite dirty. They have measured concentrations of sulfate pollutants in those cities and they have serially examined lung function in children and in adults.

The Harvard researchers have found is that exposure to acid air pollutants, especially to fine particulates and sulfates, causes increased respiratory disease among people in these cities. Children, for example, have increased bronchitis as concentrations of particulates rise in the air. Furthermore, the researchers point out that although children with a past history of asthma and wheezing are clearly the most susceptible to these effects, the effects are not restricted to those children. In fact, they are seen across the board to a greater or lesser extent in all children.

Similarly, there have been a series of studies conducted in Canada over the past decade, mostly in Ontario. These studies have monitored concentrations of sulfate and ozone in the air and have plotted those concentrations against the frequency with which children are admitted to hospital for respiratory disease, as well as for other conditions.

The Canadian researches have found that in the summer months, as levels of ozone and sulfates rise, hospital admissions in children have increased dramatically and reproducibly year after year for the past eight years, such that at certain times of the year as many as 44 percent of hospital admissions in children in southwestern Ontario are caused by air pollution.

Studies have also been conducted among children in summer camps, both in Canada and in the eastern United States. These studies have shown consistently among children in summer camp that elevated levels of ozone and elevated levels of sulfate are associated with decreased pulmonary function, and with increased frequency of respiratory symptoms, particularly wheezing and bronchitis.

Furthermore, in two of these studies, one in Canada and one in New Jersey, investigators had the opportunity to be following children along when suddenly and unexpectedly episodes of summer smog developed, with high concentrations of sulfate and ozone. Such episodes typically occur across the United States during the summer months.

The researchers observed, during those smog episodes that particularly sharp decrements in pulmonary function occurred in the exposed children. But perhaps more importantly, they observed that those decrements in function persisted for as long as a week after the episode had ceased, when the air levels were normal.

In other words, these studies makes the point that these changes in children's lung function are not just transient events that are here today and gone tomorrow. In fact, exposure to a few hours of severe air pollution at current levels can cause effects that last for at least a week.

In summary, these epidemiologic data indicate clearly that current levels of atmospheric pollution, particularly ozone, sulfates, and particulates, are capable of producing deficits of pulmonary function in children. They indicate further that these deficits can persist for a period of time.

These epidemiologic results are further corroborated by the results of studies that have been conducted on human volunteers in so-called "controlled chamber" studies. They are corroborated additionally by the results of animal experimentation where animals have been exposed to acid fog under test conditions.

The details of those studies are presented in my testimony. I will not go into them here. Suffice it to say that the results of the epidemiologic studies, the human volunteer studies, and the animal studies are quite convergent, they corroborate each other, and they all demonstrate that acid air pollution is capable of producing pulmonary dysfunction, bronchial constriction, and wheezing.

In summary, then, all of the data which have been produced in the past five to eight years in this country, and in Canada, on the effects of current levels of acid pollution indicate that these current levels can produce toxic effects in the lungs.

These are not benign effects, I submit, nor, as some contend, do they represent some form of adaptation of the respiratory tract to pollution exposure. These are, in fact, pathologic effects. Whether they actually will have long-term consequences remains to be seen. Additional research is going to be required to define whether there are delayed effects. Further research is going to be required also to determine the precise levels at which these adverse effects occur.

However, it seems reasonable to conclude at the present time that, even pending the results of additional research, two levels of control ought to be applied. First of all, there ought to be control at the source. Strict limits should be applied on the emission of sulfates and ozone from both stationary and mobile sources. Secondly, the evidence seems overwhelming that the National Ambient Air Quality Standards for ozone and for sulfates ought to be made more stringent.

I concur with my colleague from the American Lung Association that there is ample basis for the imposition of short-term ambient air standards. It has, after all, been short-term peaks in pollutant exposure that have caused most of the effects that have been observed in epidemiologic studies.

I would argue that in the present circumstances, the EPA cannot afford to wait, possibly for several or more years, for more precise research data on the adverse effects of acid air pollution on the lungs. Good qualitative data and good semi-qualitative data are available today. Considerations of cost and benefit pale, I think, when they are applied to the health of children. Any parent who sat by the bedside of a child with asthma would hardly consider cost-benefit issues.

The task confronting the regulatory agencies today is to exercise their mandate to protect not just the environment, but also the public health. I would argue that to await the completion of additional research before taking preventive action constitutes poor medicine, and it would constitute poor public policy.

Senator MITCHELL. Thank you very much, Doctor.

Before going to questions, I would like to recognize the presence of two Members of the Committee. The former chairman of the full committee, one of the great protectors of the American environment, and responsible for much important legislation enacted in the last several years, Senator Stafford.

OPENING STATEMENT OF HON. ROBERT T. STAFFORD, U.S. SENATOR FROM THE STATE OF VERMONT

Senator STAFFORD. Thank you very much, Mr. Chairman. I am very grateful for your kind words this morning.

I regret, particularly, Dr. Narkewicz, I wasn't able to get here in time to hear your testimony. I might have done better, but the State of Virginia apparently believes that the cost efficient way to get rid of the ice and snow is to wait until the sun removes it, and that sort of delays our getting in here.

Mr. Chairman, since the highway bill is under consideration on the Senate floor, I am going to be very brief, but I do have a very short opening statement.

When the acid rain debate began in earnest seven or eight years ago, there were many who said that public health was a "make wait" issue in the context of acid rain. In a sense, they were right. We really didn't know that much about the adverse health effects of acid rain.

Today we know more, I think, much more. We know enough to make it clear that protecting public health is the first reason for adopting an acid rain control program, not the last. Today there can be little doubt that there is a correlation between the acidity of the air that people breathe and effects on the respiratory tract, and these are serious effects, not minor ones.

Hospitalizations increase at the same time as air pollution, and specifically as acid rain pollution increases. In Japan when the acid rain pollution increased, so did death from lung disease, and when it went down, the death rate from bronchial asthma and bronchitis did the same.

Recent studies in the United States, Japan, Canada all point in the same direction. When acid based air pollution increases, children become sick more often and for longer periods. Children's lungs become smaller and do their airways. What this means for them in adulthood is unclear, but it seems unlikely to be favorable.

Mr. Chairman, I believe that we will hear this morning and have heard evidence that will greatly concern us all. I believe today's hearing will make clear the need to look into these studies and others in greater depth.

I have taken my share of time, but I do hope that we will have another opportunity to examine these issues before we move to markup acid rain legislation

Mr. Chairman, I thank you very much.

Senator MITCHELL. Thank you, Senator Stafford.

We are also pleased to welcome one of our new Members, the former distinguished Governor of Florida, now the Senator from Florida, Senator Graham.

Do you have an opening statement, Senator?

OPENING STATEMENT OF HON. BOB GRAHAM, U.S. SENATOR FROM THE STATE OF FLORIDA

Senator GRAHAM. Thank you very much, Mr. Chairman.

I want to express my appreciation to you for holding these hearings on an issue of such importance to our country. Having recently joined the Committee, it is of particular benefit to me to hear witnesses explain the correlation between exposure to acid rain pollutants and public health. This is an issue that has received focused attention in the past. Continued and new research in this area should prove helpful in formulating national policy.

I am particularly interested in the health aspects because so little has been said about them. Many hearings, at both the Federal and State levels, have been held on the adverse effects of acid deposition on our lakes and streams, and the damage done to aquatic life in them.

Today, we are focusing not on the threats to fish, but on the adverse effects on humans. In particular, children, the elderly and other sensitive populations appear to be at risk. Bronchitis and asthma are the primary cause of hospitalization among children under 15 years of age. Since exposure to pollutants, such as sulfur dioxide, exacerbates these respiratory diseases, air pollution is a major health issue requiring our focused attention.

Since this Committee will be considering revisions to the Clean Air Act, including the problem of ozone nonattainment, health issues must play a central role in our deliberations. I am pleased that the Subcommittee has chosen this issue for its first hearing on acid rain and I look forward to hearing the testimony of our witnesses this morning.

Thank you very much, Mr. Chairman.

Senator MITCHELL. Thank you very much, Senator Graham.

Under the rules of the Committee, we will now proceed to questioning in the order of appearance by the Committee Members, with the first round being of five minutes each, and then we will continue for as many rounds as are necessary.

I would like to ask each of the panelists to respond to this question. You can respond in the order that you testified.

Do you believe that more stringent controls of emissions of sulfur dioxide and oxides of nitrogen are warranted now based on health considerations?

Dr. NARKEWICZ. I would speak in the affirmative. I do believe that this is necessary.

What I tried to point out in my testimony, Mr. Chairman, was the particular vulnerability of the children of this nation to this threat to their health, and as usual, because of their small size, because of their inability to respond as vigorously as adults, they are indeed the focus of this environmental pollutant.

We have heard testimony by Dr. Landrigan on this panel, which clearly pointed out that even at the present levels, the problem is persisting, and it is one of the most serious disease caused environmental situations that is responsibile for 21 percent of the hospitalizations in children under 15 years of age.

Yes, I do believe that this is a serious problem and it should be addressed.

Senator MITCHELL. Dr. Godar.

Dr. GODAR. Mr. Chairman, the American Lung Association has in the past taken the position that unless we can document health effects associated with acid rain that, although we are not in favor of the death of fish and trees, to maintain what we view as our scientific credibility, we do need to become involved in the issue if and when we feel there is truly a health impact for the human population.

We have no doubt, nor had we any doubt, that precursors of acid rain are, indeed, hazardous. As a matter of fact, in the past, they have been major causes not only for morbidity, but for mortality. I would point out that the death of the human is a rather severe end-point when one looks at cause and effect.

Morbidity is one thing. If the pediatric population has increased incidence of respiratory symptoms during childhood as a result of a pollutant such as sulfur oxides combined with particulates, and particularly with ozone, then let us consider that when that child grows up, the incidence of respiratory disease in adults will be increased, and the health care cost of that will be magnified.

It is very clear that when you have respiratory disease in children, you have respiratory disease in adults. We don't understand all of those mechanisms, but it means that the investment in safety for the pediatric population means investment in cost reduction for the health care in the adult in a very, very direct way.

Therefore, as far as the American Lung Association and its scientific assemblies are concerned, precursors of acid rain are, indeed, significant causes for health effect. We feel there is a correlation between acid rain and health effects.

Senator MITCHELL. Do I take this as an affirmative answer?

Dr. GODAR. Yes.

Senator MITCHELL. You do believe that controls on emissions are warranted based upon health considerations.

Dr. GODAR. They are, indeed.

Senator MITCHELL. Dr. Robbins, would you respond to that question please?

Dr. ROBBINS. The answer from the American Public Health Association is very clearly yes. The association has consistently taken that position. What we have now is very clear evidence that the very same pollutants that produced the phenomenon that has been seen as a problem for landscape and the environment is now a problem for human health. On that basis, the control of emissions are warranted.

Senator MITCHELL. Thank you.

Dr. Landrigan.

Dr. **Landrigan.** I, too, answer in the affirmative, Mr. Chairman. The recent epidemiologic evidence, the clinical evidence, and the laboratory evidence, all indicate that current levels of acid air pollution are responsible for increased respiratory disease in children. They may additionally be responsible for increased risk of chronic lung disease in those same children as they grow to adulthood. I think that these data constitute compelling reasons to lower existing standards.

Senator **Mitchell.** Thank you.

I think that this is a significant statement that I hope is noted beyond this hearing room, that here we have the four positions, the President-Elect of the American Academy of Pediatrics, the President-Elect Designate of the American Lung Association, the Past-President of the American Public Health Association, and the Director of Environmental and Occupational Medicine at Mount Sinai Medical Center in New York unanimously recommending to this Committee or advising this Committee that in their professional opinion, based upon their experience, that health considerations now represent a sufficient basis to justify legislation or action controlling emissions of sulfur dioxide and nitrogen oxide.

That is a significant development. Heretofore, the debate has focused entirely upon physical damage to our Nation's waters, which has been clearly demonstrated, and potential damage to our Nation's forests. This adds an important dimension to the debate on acid rain control. We thank you, gentlemen, for your testimony.

My time is up on the first round of questioning.

Senator Baucus.

Senator **Baucus.** Thank you, Mr. Chairman.

I agree that this is an important statement that ought to be made, and I would like to follow up on that.

In your professional opinion, what is the best argument against your conclusion?

Secondly, what is the best offsetting argument so that on balance you think that, yes, there is enough evidence that sulfur dioxide and nitrogen oxide, for example, as precursors to acid rain, do have sufficient adverse health effects in order to warrant legislation?

Let's go down the line here. What is the best argument against your conclusion, as a professional, and what is the best offsetting argument?

Dr. **Narkewicz.** I think that the best argument against what we have stated is that there is not enough specific literature available to make every point possible that each and every one of the pollutants that we talked about this morning have been researched in detail and that we have absolute, positive, compelling proof that under any given circumstance that by reducing it, you are going to reduce the effect.

When in truth the argument should be that air pollution is such a complex issue because, as I tried to point out in the testimony, the dose, the duration, the combination of events taken together, have an effect which may be more cumulative than each and every individual components that the people in the laboratory are studying.

I say on balance, then, that the weight of the data would argue that it is a serious problem, it is an increasing problem, and that we, as citizens of the United States, should take some action on this issue.

Senator **Baucus**. Dr. Godar.

Dr. **Godar**. Senator, the best argument that one could mount might be that if you were perniciously interested in population control that that was a form of population control.

Senator **Baucus**. I want a nonpernicious argument.

Dr. **Godar**. It is often argued that when one looks at biological material that some people seem not as affected as others and, therefore, perhaps, there is not a true problem. That is also true for lung cancer and smoking. The point is, we have genetic variation, and some people have more resistance and others have less to the pollutants.

We are looking at data where some seem to have less effect; they are perhaps more sedentary, and they have less hyper-reactivity. That sort of negative data suggesting that not all are uniformly affected is often used as an argument. It is a totally spurious argument.

It is just like the study of any phenomenon in biological material, there is always a spectrum of effects from severe to none, and the mean effect is highly significant and affects the majority of the population, that is, the curve is very broad-based in this particular case. I don't really feel that there is a significant argument.

Senator **Baucus**. Dr. Robbins.

Dr. **Robbins**. I would concur that there is no significant argument. You will hear many specious arguments.

Senator **Baucus**. Let's be honest about this. There will be some who will come in here and say, there is not sufficient data, and some may legitimately take that view. So I am asking you, as a professional, to give your best argument, and then on balance why you think that this is not the way.

Dr. **Robbins**. You will hear the argument that there are insufficient data. I believe that the tobacco companies are still saying that there are insufficient data in terms of cigarettes causing cancer.

I was going to say that the thing that you may also hear, which is irrelevant, but probably correct, is that more damage is being done by cigarettes in this country and tobacco than this air pollution, but I don't think that this is in any way a reasonable basis for not acting on this problem.

Senator **Baucus**. Dr. Landrigan.

Dr. **Landrigan**. I think that the most plausible argument that will be raised against lowering the standard is that there are insufficient data to formulate a clear dose response relationship that relates a particular level of pollutant in air to a particular frequency of disease in exposed people.

It is a truism in toxicology and epidemiology, and in medicine generally, that the greater the exposure of a person or population to some toxin, the more frequent and the more severe ought to be the resultant disease.

In the case of acid air pollution, we do not yet have absolutely precise data which enable us say with total certainty what particular level of ozone is associated with a particular frequency of asthma.

Some critics, therefore, will argue that in the absence of that sort of data out to the third decimal point, we ought to postpone setting any more stringent standards or passing any new legislation, that we ought to wait a few years, that we ought to be "completely rational" about the process, until we have further research in hand.

I think that that is a bad argument. Although it is true we do not yet have answers out to the third or the fourth decimal point, we certainly have pretty good semiquantitative information, sufficient to show the adequacy of the present exposure standards.

Senator BAUCUS. That is essentially Dr. Narkewicz's point, that is, we haven't dotted all the "i's" and crossed all the "t's", but the cumulative data is overwhelming.

Thank you very much, I appreciate it.

Senator MITCHELL. Thank you, Senator Baucus.

Senator Chafee.

Senator CHAFEE. Thank you, Mr. Chairman.

Do each of you gentlemen agree that the studies cited by Dr. Landrigan are the strongest cases to substantiate the point that you are making? In other words, are these the best studies that you know of?

Dr. NARKEWICZ. They certainly are some of the best studies that I am aware of, but that is not to discourage any further studies that might shed some light on this important issue.

Senator CHAFEE. There will be plenty of enthusiasm for more studies, you don't need to worry about that.

Dr. Godar.

Dr. GODAR. I think that is true and I would point out that I had the pleasure of being present when Dean Shepherd from the University of California at San Francisco presented his data on asymptomatic, relatively healthy young asthmatics, truly a limited study in terms of the number of volunteers, but the effects of sulfur dioxide were remarkable at very low levels and with modest exercise. This effect was potentiated by exposure to cold.

Senator CHAFEE. Which study is that, Doctor, and was it cited by Dr. Landrigan?

Dr. GODAR. That study is not part of the Six Cities Studies, but it is cited in the literature, in the American Review.

In that study, he pointed out to the administrators of technical groups that he was really talking about highly selected, asymptomatic people known to have asthma, but currently having no problems. I was personally impressed at the low level and the speed with which the decrement in function and the symptoms began. In fact, in some cases, within two minutes of beginning the study.

Senator CHAFEE. Dr. Robbins, if you were called upon to substantiate your points, would you cite these same studies as Dr. Landrigan?

Dr. ROBBINS. Dr. Landrigan and I are both aware that there have been some recent reviews of these studies conducted at the National Institute for Environmental Health Sciences. I would guess that that list of studies will be available to you in the future, and it is probably the most up to date list. It may be slightly more extensive.

Senator CHAFEE. What are those, Doctor?

Dr. Robbins. There was a workshop held earlier this year at the National Institute for Environmental Health Sciences where all of the relevant studies on these issues of acid rain and health effects were reviewed.

I have only seen a very preliminary summary of that meeting and the studies that were reviewed, and I have not reviewed all of those studies at this time. I think that it would constitute for the committee a reasonably thorough review and list of the relevant studies.

Senator Chafee. Are there any studies that could be cited by the other side as just disproving these studies, Dr. Landrigan?

Dr. Landrigan. Yes, there are a number of studies that have failed to show correlations between levels of air pollution and respiratory effects in children. There is a study, done a couple of years ago in New York City, for example, at Columbia Presbyterian Hospital, by Dr. Inge Goldstein and some colleagues, which failed to show a correlation. Those studies will undoubtedly be cited to you.

However, I think that it is an established scientific principle, in a difficult area such as reactions of respiratory tract, that positive studies ought to weigh more heavily in evidence than negative studies. Let me explain why that is so.

Senator Chafee. Let me just say why I asked the question. We have to be forewarned because there are people who feel very strongly on the other side of this issue, and they will cite, possibly, this study from Columbia Presbyterian Hospital, was it?

Dr. Landrigan. Yes, sir.

Senator Chafee. What is wrong with that study, why don't you continue?

Dr. Landrigan. The point is there are many factors that influence the susceptibility of children to asthma. Children differ genetically. A child who is having a cold or a viral infection this week is going to be more sensitive than the child who is not having an infection. A child who is exposed to dust in the home, for example, cat dust, or dog dust, is going to be more likely to have an asthmatic attack than a child who is not so exposed.

Air pollution is only one of a number of factors that combine to produce attacks of asthma and episodes of bronchitis in children. In other words, there is a lot of background noise, i.e., a low in the signal to noise ratio. It is not surprising, then, that not all studies fail to discern a signal. Some are going to miss it.

Senator Chafee. Why should we believe your studies, then?

Dr. Landrigan. I think that what you have to do, you have to weigh the studies one at a time. You have to look at them, and you have to compare the methodology. You have to examine how carefully they look at various factors that may confound and complicate the results.

One of the strongest arguments that I would put forth for accepting the studies that I have cited is the fact that different studies, conducted under different circumstances, at different times, and in different populations, have all corroborated each other.

There have been epidemiologic studies in eastern Canada, in central Canada, in Pennsylvania, in New Jersey, the chamber studies in San Francisco, and toxicologic studies at Rochester, all of which have come up with fundamentally the same results.

That sort of consistency across six or eight different studies constitutes one of the best arguments for the fact that you are seeing a real problem.

Senator CHAFEE. Even though the Columbia study shows otherwise?

Dr. LANDRIGAN. That is all right. Not every study that has examined the relationship between the exposure of children to lead and the subsequent development of mental retardation has discerned the effects. Some studies have been less well done than others, or they have measured the wrong thing at the wrong time and haven't seen the effect, and yet the concept that a relationship exists between lead exposure and mental retardation is now almost universally accepted among pediatricians.

Senator CHAFEE. Thank you, and thank you Mr. Chairman.

Senator MITCHELL. Thank you, Senator Chafee.

Before proceeding with the questioning, I would like to recognize Senator Durenberger, a valuable Member of the Committee, who arrived during the questioning.

OPENING STATEMENT OF HON. DAVE DURENBERGER, U.S. SENATOR FROM THE STATE OF MINNESOTA

Senator DURENBERGER. Mr. Chairman, I thank you, and I beg the indulgence of my colleagues for a brief opening statement.

I want to compliment you and the witnesses who have come here today on short notice. Last year, when we had these hearings, the then Minority Leader on the Democratic side had one of those horrible things happen to him; one page of his testimony wasn't there. Some of us who were here remember that horrible experience; it is one of those things we joke about on how staff could get even with us. That staff member is probably no longer employed by the Senator from West Virginia.

This year, he is the Majority Leader. He spoke yesterday at the American Mining Congress about his opinion, and he stands about where the Chairman of the Energy and Commerce Committee on the House side stands on some of these issues. So I compliment you, as a person who is generally credited with having performed some minor miracles, we think they are minor.

The gentleman on my left and your right, Mr. Chairman, probably has been at this much longer than we have, and each of us lives each year in the hope that this might be the year where some legislation reaches the President's desk. I want to be an optimist, and I want to hope that these four gentlemen, some of whom have been here before, that their trip today is not in vain.

I would like to make one brief point this morning, if I might, that isn't just on the linkage between acid rain and the health effects of the precursor pollutants, but I think that some of our witnesses this morning, I am told, have pressed on the committee their view that ambient air quality standards, under the Clean Air Act, need to be amended to include more stringent ambient standards.

Although each of these may not be mentioned specifically by each of the witnesses, I have certainly seen evidence suggesting a need for a tighter one-hour standard for sulfur dioxide, the addition of standards for sulfates and fine particulates, and a tightening of the existing ozone standard.

I would like my colleagues on the Committee to know that I agree with the witnesses on those views. I think that the ambient standards need to be tightened more than ever. I believe that we should consider reaching these objectives legislatively by mandating these steps through amendments to the Clean Air Act.

Acid rain legislation has been introduced by the distinguished Chairman of the Subcommittee, Senator Mitchell, and the Ranking Member of the Full Committee, Senator Stafford. Both bills would require extensive reductions in the emission of acid rain and ozone precursors, principally sulfur dioxide and nitrogen oxides, but neither would amend the Clean Air Act to require the modifications for ambient sulfur dioxide, sulfate, particulate, and ozone standards.

I don't mean to criticize either bill by raising the issues because I am a cosponsor of both. I don't think that either of these bills were intended to preclude legislative options on ambient standards. I suspect that the authors simply chose not to address those questions directly in the draft of acid rain bills. But I think we should address those questions directly at some point in the process.

Everyone acknowledges, I think, that the bill reported by the Committee will include more than the acid rain controls. We must deal with the nonattainment question. I will personally press the Committee to consider air toxics amendments, and I am sure that other Members will raise other issues.

As we consider those other questions, it will be important to keep in mind the status of the ambient air quality standards that EPA has developed and to ask whether modification of those standards is appropriate.

For instance, I believe the scientists will tell you that the evidence indicates that the ozone standard should be brought down to at least 0.08 part per million. If we agree, and I happen to, that is certainly that we need to take into account when addressing other non-attainment problems.

My State of Minnesota is a perfect example of the implications. We are in compliance with the ozone standard now. Some areas of the State would not be in compliance with the standard of 0.08 parts per million. I can't come to judgment on a nonattainment without considering the possibility that the standard will be lowered.

I am sure that a tighter, short-term sulfur dioxide standard, or the addition of a sulfate standard would also have implications for the kind of acid rain control program that we design.

That is the points that I wanted to make this morning, Mr. Chairman, together with expressing my appreciation to you for your true grit, and express my commitment to you and the other Members of this Committee who would like to make the 100th Congress the year in which we do attain, perhaps through the Clean Air Act or somewhere else, a meaningful legislation in this area.

Senator **MITCHELL**. Thank you, Senator Durenberger.

Senator Stafford.

Senator **STAFFORD**. Thank you very much, Mr. Chairman.

First let me join in expressing my appreciation at your scheduling these hearings and my gratitude for all of the yeoman work you have done in past Congresses to allow us to do most of the environmental issues we felt we wanted to do. We could not have done them without you, Mr. Chairman.

Senator **Mitchell**. Thank you.

Senator **Stafford**. I say that also to the Senator on my right, Senator Durenberger, who was so instrumental in some of the major bills this committee produced.

One piece of legislation that escaped both the years when Senator Randolph was Chairman of the Committee and the years that I had that privilege, was acid rain legislation. It certainly has one of the highest priorities as far as the Senator from Vermont is concerned.

In listening to the witnesses respond to questions, having already apologized for not having arrived here on time, I do suggest, Mr. Chairman, that you consider and that the committee consider that there be more hearings on the matter of health effects as far as the various aspects of acid rain are concerned.

I would also suggest that arrangements could be made with the appropriate Government agencies to assemble a brief of about 20 pages, a summary of recent advances in air pollution health effects from their viewpoint.

I would finally ask the staff be instructed to review the fiscal year 1988 budget to determine what money is being made available for health effects research and, if appropriate, prepare recommendations for increases as far as the budget is concerned.

I would simply make those suggestions, Mr. Chairman, for your subcommittee to consider.

I also want to welcome Dr. Robbins here, another distinguished representative of the State of Vermont in past years. We are glad that you are here, Doctor. It is nice to see you again.

Let me ask this very brief question of all of you. Is there any question in your minds, individually, that acid rain and its associated pollutants pose a significant health problem to humans, especially those sensitive populations such as children and asthmatics. It almost calls for a yes or no answer.

Dr. **Narkewicz**. It would under circumstances, Senator Stafford, but since everyone is in a complimentary mood, I would like to take this opportunity and compliment the committee for its interest and concern, and in particular that you would invite a pediatrician and the American Academy of Pediatrics to hear views on children, partly because they don't vote and because they don't contribute to the coffers of those who represent us. So we are particularly happy that you have invited us because we do feel that we are advocates for the children, and we think that you are also.

I would answer very definitely yes.

Senator **Stafford**. Dr. Godar.

Dr. **Godar**. I have no doubt of the relationship. I would point out that the Lung Association's constituency does include the pediatric age group, the people with lung disease and, in addition, those over 65. For those who have reached 65 or are approaching it, we are the vulnerable group.

Senator **Stafford**. Dr. Robbins.

Dr. ROBBINS. I also have no doubt, and thank you, Senator, for all the work that you have done in this area.

Senator STAFFORD. Dr. Landrigan.

Dr. LANDRIGAN. I answer yes.

Senator STAFFORD. Thank you, gentlemen.

Is it fair to say, in view of your answers, that the health effects of acid rain will occur wherever there is air pollution?

For example, my recollection is that the highest rate of acidity, or the lowest Ph factor in acid rain ever recorded occurred in West Virginia. I would assume that the health effects, therefore, would flow in West Virginia and to other Midwestern States as fully as they might in the Eastern Seaboard and the Canadian maritime provinces where acid rain has fallen. Am I correct in that?

Dr. NARKEWICZ. Yes, I think, you are, except if I might make a point about children, and particularly about asthmatics.

I think that it is difficult to point up exactly why that might occur because when you think about the diseases that we are talking about, asthma and bronchitis, the process that occurs is that the pollutant acts as a stimulus or a catalyst to whatever is going to happen in the lung, and that catalyst is something that may be there for only a short period of time, and for that particular person, given a variety of other circumstances, it may not be as high as is necessary in some other part of the country.

Once the process starts, it is almost irretrievable, and the lung has a certain process, and I pointed out that children are more susceptible to that process. So while you may be correct, I think, that in general the problem is a national problem, although sometimes it may be a local problem in some other part of the country.

Senator STAFFORD. Dr. Godar.

Dr. GODAR. Senator, I think it is true, however, contingent upon the fact that concentration and duration of levels vary widely. For many people, unless they see bodies on the sidewalk, they really don't feel that there is a significant correlation.

I am particularly concerned about ozone because the entire State of Connecticut is not in compliance. As a matter of fact, although we have made improvements as a result of inspection and maintenance, and although we do import ozone from other areas, pointing out that many of these pollutants are regional and not local, they require a coordinated effort.

But I am impressed that I hear from patients, rather independently, without coaching, that as they take their weekends in Vermont or New Hampshire, for reasons beyond me because I think Connecticut is a lovely State, but they seem to be more comfortable in Vermont, Maine and New Hampshire, and as they are returning on a Sunday to Connecticut, the symptoms begin recurring as they reach the Massachusetts border and intensify when they reach Connecticut.

I am, therefore, convinced that I see in clinical practice, my own little mini-study, not well controlled, very significant effects on individual patients I know well that seem to correlate with the geographic zone and with ozone levels during the summer months.

I also note that being from Connecticut, I am one of the few people here not from Vermont, and I apologize.

Senator STAFFORD. Doctor, it is too bad that we can't pipe the ozone at the troposphere level into the stratosphere where we need it.

Dr. Robbins.

Dr. ROBBINS. Senator, I don't know whether you envisioned in your question the problems of workers. To the extent that the Middle-West and the Appalachian area, with its population of coal miners exposed to dust, and other industrial workers in foundries who are exposed to silica, and have developed pneumoconiosis, these are a separate susceptible population that is going to be more sensitive to comparable levels of air pollution. In that sense, it may in fact be just those areas that have additional populations at high risk.

Senator STAFFORD. Thank you.

Dr. Landrigan.

Dr. LANDRIGAN. One of the studies that was conducted, to which I referred to, was undertaken in a summer camp in western Pennsylvania, in the Town of Indiana, Pennsylvania, which is right near the West Virginia border, near Pittsburgh. It showed a distinct relationship between ozone concentrations and changes in lung function among the children in that camp.

I think therefore that it is fair to say that these events extend to the Midwest.

Senator STAFFORD. Thank you.

My time is up, but Dr. Robbins anticipated the last question I had.

I would say to my friend, Senator Durenberger, that in the Mitchell and Stafford bills, we do attempt to deal, to a degree, with the ozone question, because we recognize it as a very serious one.

Senator MITCHELL. Thank you, Senator Stafford.

Before proceeding with the questioning, I would like to recognize Senator Pressler, who has just come in, for an opening statement.

OPENING STATEMENT OF HON. LARRY PRESSLER, U.S. SENATOR FROM THE STATE OF SOUTH DAKOTA

Senator PRESSLER. Thank you, Mr. Chairman. I will place my opening statement in the record, except to say that I apologize for not being here this morning. We are having the first hearing on the budget of NASA over in the Commerce Committee, of which I am the Ranking Member.

I thank you for holding these hearings. Indeed, my State of South Dakota has a better situation than some, but we are even finding increasing air problems. Indeed, I think that South Dakota has been found to be affected by acid rain, so the problem is coming.

We read about lakes and streams being polluted. Often our initial response is that we should not drink that water or eat fish from it, or we are thankful we don't live there. Unfortunately, people do think about the fact that they are often exposed to the same elements that have polluted those lakes and streams.

Acid rain and other air pollution issues are not regional issues, but they have become international issues, certainly national. Pollutants have been traced from Europe to the Arctic Region. This illustrates that these issues are no longer domestic issues.

Today, we are examining the health effects of those pollutants, and I have read some of the testimony and find it very startling. Will these pollutants have the same effect on humans as they have had on fish, wildlife and plants, that certainly seems to be the case.

Our people are being exposed to sulfur dioxide in increasing amounts, nitrogen oxides, sulfates, nitrates, and many other pollutants every day. Several of these pollutants are not regulated under the Clean Air Act.

Should standards be set for all these pollutants, and how are the current and new standards to be met?

Does this exposure have a cumulative effect on people, and are certain people with allergies or asthma adversely affected?

These questions have not only environmental implications, but also human health implications, and also cost to the Government. I just came from a meeting with a group of people from our hospitals in South Dakota, and they were talking about the cost of medical care.

Certainly, if we are going to be a productive society, if we are allocating large resources to problems that result from air pollution, it will be very costly, aside from the human factors involved.

Mr. Chairman, I have questions, but I shall wait until my turn. If I have to leave, I will submit them for the record.

[Senator Pressler's statement follows:]

OPENING STATEMENT OF HON. LARRY PRESSLER, U.S. SENATOR FROM THE STATE OF SOUTH DAKOTA

Mr. Chairman, I want first to thank you for scheduling this hearing on the health effects of acid rain. In the past, environmental impacts have been the primary focus of acid rain hearings. For some reason, when we talk about fish or trees dying, people do not get as concerned as when their own health is affected. This hearing is critical in focusing our attention on the human health implications of air pollution.

People read about lakes and streams being polluted. Often the initial response is that we should not drink that water or eat fish from it. Or thank goodness we do not live in that area. Unfortunately, people do not think about the fact that they are often exposed to the same elements that polluted those lakes and streams. Acid rain and other air pollution issues are not regional issues and have become international issues. Pollutants have been traced from Europe to the Arctic region. This illustrates that these issues are no longer domestic issues. Today we are examining the health effect of these pollutants.

Will these pollutants have the same effect on humans as they have had on fish, wildlife and plants? That is one issue I hope will be addressed today. People are exposed to sulfur dioxide, nitrogen oxides, sulphates, nitrates and many other pollutants every day. Several of these pollutants are not regulated under the Clean Air Act. Should standards be set for all of these pollutants, and how are the current and new standards to be met? Does this exposure have a cumulative effect on people? Are certain people with allergies or asthma adversely affected? These questions have not only environmental implications but also human health implications. These relationships must be carefully studied to permit Congress to effectively address the issue of air pollution and to enable the medical profession to effectively treat the potential health effects.

I look forward to hearing the comments of the witnesses.

Senator **MITCHELL**. Thank you, Senator Pressler. You will not have long to wait for your turn.

Senator Graham, you are next.

Senator **GRAHAM**. Thank you, Mr. Chairman.

I am interested in trying to put this public health issue into a relative context of other public health issues. Could you assess how you would rank this in terms of the types of environmental and public health concerns which we, as the Congress, should be directing our attention towards?

Is this in the upper 10 percentile of concerns for public health in America?

Dr. NARKEWICZ. I find this difficult to answer since I am a practicing pediatrician, and not a researcher or anything else. But as far as children are concerned, I can just say that it is in one of the uppermost categories of concern.

I think accidents are perhaps one of our top concerns above that, but certainly environmental pollution, which may have a very long term effect on the children of the country ranks very high.

Dr. GODAR. Senator, I have difficulty giving it a ranking, except to say that the precursors to acid rain have already been demonstrated to kill people. Accordingly, this is simply one form of pollution by two or three agents known to be hazardous to man and, therefore, whether we approach them as individual pollutants or as the product of combined pollution leading to acid rain, we are really only talking about form.

Essentially, sulfur oxide and particulates were the major reason for large numbers of deaths in the disastrous pollution episodes of decades past. Experimental animals have been affected by sulfates and particulates for as long as they have been used as experimental animals for health effects.

There is good correlation between the level of sulfur oxides in the community and the overall mortality rate from all causes, so the data comes from all directions. So acid rain and its precursors represent one part of a large picture. In that respect, yes, these are important pollutants. I don't know where to rank the acid rain problems specifically as a component, it is very difficult to do.

Dr. ROBBINS. Senator, it is a difficult question. As the information comes in and the health effects of this kind of pollution become clearer, I think we can and will get some reasonable answers about the number of deaths, and what is happening.

It is probably less of a problem than smoking. It may be less of a problem than occupational exposures to toxic materials. But it is probably more of a problem than many of the other things that we are concerned about in society.

I think the thing that is important to recognize in relation to this pollution is that we do know how to reduce it. It is terribly important, when one is thinking about public health, to consider the solutions, and that they are available. Here one is available through reducing these emissions. So it is a solvable problem, and whenever we have death and disease caused in a manner that is preventable, it puts it higher on our list than some of the problems that we do not, right now, know how to solve.

Dr. LANDRIGAN. Dr. Robbins and I worked together for many years, so my conclusions are fairly similar to his. There is no question that smoking is the major cause in this country of respiratory disease.

Active smoking is number one, and passive smoking is probably number two. Then occupational exposures to lung toxins are a major and preventable cause of disease. I think that acid rain comes third after those. But I would amplify a bit on what Dr. Robbins said about the preventability of acid rain, and I would argue that it ought to be possibly number one on your agenda because of those three leading causes, it is the one that is the most susceptible to regulatory and legislative prevention.

There is a limit to what you, as Senators, can do about people's desire to smoke. With regard to occupational exposure, you have good laws on the books, but you have the problem of having to enforce those laws through hundreds of thousands of workers across the country. That is obviously a mind boggling task even if OSHA were up to it.

But in the case of acid rain, there is really a finite number of major sources of acid air pollutants. They can be targeted with relative ease. In fact, they have been pretty well targeted already by the EPA. A few swift and sure legislative strokes could deal with those sources.

Senator **Graham**. My time is up, Mr. Chairman. Thank you.

Dr. **Godar**. May I add a comment to the answers. With cigarette smoking, that is a personal choice. If one wishes to commit suicide in that manner, one has the personal choice of so doing. The problem is that it is a little bit like passive smoking in that you don't have a choice, you simply happen to be in the wrong place. I think that our citizens are entitled to protection against something for which there is acknowledged protective mechanisms.

So for those who choose not to be occupied in a dangerous occupation, where they are given some form of hazard pay and tacitly accept this and the risk, or those who choose to smoke and accept the very high risk, in this case, they have not made that choice, and they really have no choice.

Senator **Mitchell**. Thank you, Senator Graham.

Senator Pressler.

Senator **Pressler**. Let me ask you, how much of this is new in terms of health? Didn't people living in London in Charles Dickens's time have a much worse situation, in fact?

Dr. **Narkewicz**. Like the children who always have to go first, I feel I have to go first.

How much of this is new, certainly in the Mews Valley Region in 1930, the huge numbers of people that were killed and injured with a single environmental pollutant was very dramatic. In 1948, it has been quoted, in Donora, Pennsylvania, 13,000 inhabitants.

Those were dramatic episodes, and it focused people's attention on it. But now, when we finally have the evidence before us that the on-going pollution, not the dramatic effects that we read about, but the on-going pollution, is adding each and every day to the morbidity and quality of health for children. Then, it has probably always been there, and we are just now focusing on the long-term public health preventative effects of it.

Dr. **Godar**. There is no question that the major causes for death in the catastrophic events in past centuries and in past decades has, in fact, been the sulfur dioxides, so we are basically talking about the same pollutant.

I am impressed by graph after graph documenting some sort of linear relationship between morbidity in man in general and the sulfur oxides levels, between the morbidity from pollutants and urbanization. I think that the pattern is very clear. We are simply seeing another form of the same phenomenon.

Dr. ROBBINS. Senator, I would concur with the two previous witnesses. I think that the one area where, as we learn more, the evidence is very new and might not have been expected in the past, has to do with the releases of toxic materials into the environment and their effects in terms of subtle neurologic effects, in terms of cancer.

We are learning more and more about that all the time, and we are becoming increasingly concerned about air pollution in that way, just as the evidence is finally clear and solid with regard to the pollutants that are producing acid rain.

Dr. LANDRIGAN. I would second that, too. The diseases are certainly not new, nor are the pollutants, but what is new is that as levels of pollution have come down in the environment, subsequent to the Clean Air Act, we have developed new technologies and new medical methodologies which enable us to see toxic effects at levels of exposure that five or ten years ago we thought were safe. It is this increased sensitivity in scientific methodology and our enhanced ability to find disease at low levels of exposure that, I think, is driving the present discussion.

Senator PRESSLER. Dr. Godar, in your testimony, you mention that the exposure to levels of sulfur dioxide found in the ambient air combined with exposure to cold dry air is worse. Is it worse in the wintertime because of the coldness of the air, or because the air stops moving, or what?

Dr. GODAR. The cold dry air potentiates reactivity in the respiratory tract, whether it is in asthmatics or whether it is someone with hyper-reactive airways who does not yet have asthma.

I would remind the Committee that approximately 20 percent of the population are classified as hyper-reactive. Allergists call these people with twitchy airways. Their bronchial system is very sensitive. Of this 20 percent, one out of five of the entire population, 40 percent of that group at some time during their life will become asthmatic, symptomatic asthmatic.

So we are not talking about a small group. We are talking about a large constituency, and you are talking at a minimum of 8 percent of the population. I personally believe that it is a conservative estimate. Those people are, in fact, very sensitive to the same pollutants to a higher degree and are also particularly sensitive to cold dry air as a stimulus of reactivity of the respiratory tract. So it is of interest to me that there should be this potentiation.

Senator PRESSLER. Are there hard number comparisons that compare the health in parts of the country that are not so polluted? For example, North and South Dakota, Montana, are the young people there healthier?

What is the healthiest part of the country to live in, and what hard comparisons do you have?

Dr. GODAR. I think that the healthiest place to live is obviously Vermont.

Senator PRESSLER. But Vermont is right in the center of the acid rain, isn't it?

Senator STAFFORD. You are welcome there any time, Senator.

Senator PRESSLER. Isn't Vermont right in the center of the acid rain?

Dr. GODAR. It certainly is affected. The point is, if we talk about combined pollutants, which includes ozone and other pollutants, then we have a somewhat different picture, and it is skewed by season as well.

One of the major problems is the variability of sensitivity. For many years, in measuring pulmonary function tests in socalled "normal people," we have looked for what are called "normal values," which is hard to define. We are really looking for sort of average, undiseased values.

We did not have adequate values for lung function tests until approximately 1971, and the reason is that all of the studies of socalled "normal people," indeed, had not accounted for the fact that they did have some form of disease in the past.

The first study that showed data similar to normal volunteers in our laboratories were data performed by someone who studied nonsmoking, healthy populations in the West, in a very, very rural setting, and their lung function was as high as any group, and from there it was downhill. I would suggest, for example, that if you lived and were raised in New York, you might well have subnormal function simply by having been an urban child.

Senator PRESSLER. What I am getting to is, based on the diseases and the symptoms that you have described here this morning, is here a healthiest part of the country to live in?

Dr. NARKEWICZ. Senator Pressler, I would like to answer that as far as children are concerned, I can't say, but I can tell you what we think that the safest place for a child to live in the country is where there are no industrial pollutants in the air, in a loving home where the family doesn't smoke.

Senator PRESSLER. It sounds like South Dakota.

Dr. LANDRIGAN. I can't expand on that, but I can add more detail. The Harvard School of Public Health Six-City Study, to which I refer on the second page of my testimony, looked at the relationship between air pollution and health effects in six locations around the central and eastern United States, Steubenville, Ohio; St. Louis, Missouri; Watertown, Massachusetts; Kingston-Harriman, Tennessee, Topeka, Kansas; and Portage, Wisconsin.

They found that the cleanest air of those six cities was in Topeka, and the very cleanest of all in Portage. They found definitely less respiratory disease in those two clean cities. The worst two were Steubenville, Ohio—which was the very worst, which is on the Ohio River, within the line of sight of West Virginia—and St. Louis, Missouri. They found high rates of pulmonary, asthma and bronchitis, in those two places, particularly in Steubenville.

Senator PRESSLER. But there hasn't been a comparison of health statistics of a very rural area to an urban area the real hard-nose health statistics.

Dr. ROBBINS. Senator, it is not too difficult to compare death rates for groups of diseases state by state. What has always happens is that cigarette smoking is so important in these results, and Utah has consistently, with its Mormon population, come out with the lowest death rate from both cardiovascular and respiratory disease. I seem to recall, and it is a few years old, that West Virginia and some of the midAtlantic States had the highest death rates in those two areas.

What is very impressive, when you compare the states is that the differences are very large. There aren't small differences between States in terms of these death rates, but a State may have almost twice as many deaths from a particular group of diseases as another State. This is the kind of information that is easily available from the National Center for Health Statistics.

Senator MITCHELL. Thank you, Senator Pressler.

We will go to the next round of questioning now, and I will begin by asking a question related to what Senator Pressler has been asking, but in a different way.

You have described certain health effects from certain pollutants. Are these health effects limited to any specific areas of the country, or are they observable throughout the country?

Dr. NARKEWICZ. I believe that the effects of the pollutants that we are talking about are observable, if you look for them critically, in any portion of the country.

Senator, that will have to be my concluding remark because I have an airplane to catch. I appreciate the opportunity to speak before this committee.

Senator MITCHELL. Thank you very much, Dr. Narkewicz.

Dr. Godar.

Dr. GODAR. The effects are going to vary with concentration time and there may be other factors. We have not included socio-economic factors, which may make a subtle difference. But epidemiologists are accustomed to accounting for these.

There is hardly any part of the country that does not have a problem with pollution, and most of the pollutants we have discussed are not local, they are regional, they are nationwide. The concentrations may vary from zone to zone. What concerns me most is that local control efforts are not enough.

It is truly a regional problem, and as long as the wind direction is shifted constantly, it is hard to predict at what time the pollutant levels will be highest in what part of the country. Essentially, then, we are all affected to a variable degree, and we should not consider that there are any pristine areas any longer.

Senator MITCHELL. Thank you.

Dr. Robbins.

Dr. ROBBINS. I think that Dr. Godar is absolutely right, but I would add that it is the difference in pollution levels that means that places closer to the sources may be exposed in one way from the direct local effects, and then the much broader plume from such pollution spreads out causing distant and more indirect effects.

So no one escapes, but it is important to understand the different kinds of effects that you will be preventing as you reduce emissions.

Senator MITCHELL. Dr. Landrigan.

Dr. LANDRIGAN. I have nothing significant to add to that, Senator.

Senator MITCHELL. Dr. Landrigan, I would like to ask you. Does an increase in acute respiratory incidence affect resistance to other diseases?

Dr. **Landrigan**. Some of the studies that were conducted in Britain ten or fifteen years ago showed quite clearly that children who are more heavily exposed to air pollutants have a higher frequency of respiratory infections than other children.

Precisely comparable studies have not been conducted in this country. However, I think that it makes good biological sense to think that a similar pattern would appear, because what happens in asthma is that the airways constrict, mucus in them thickens, and clearance of secretion is impaired. All of those are factors which are known to increase sensitivity to infection.

Senator **Mitchell**. Dr. Robbins, you said something in your opening remarks that caught my attention, and I wonder if I could ask you to repeat it, because it wasn't clear to me. You mentioned the drinking water supply of the City of Boston, a reservoir about which you made a comment. I wonder if you would repeat that?

Dr. **Robbins**. I was picking up from Dr. Walker's prepared testimony, and from the knowledge I have gleaned since moving to Boston University. Quabbin Reservoir is a large reservoir outside of Boston. It is the major source of water for the City of Boston. It has recently been shown to be very close to reaching the end of its ability to buffer the acidity that comes in the form of rain, and at that point there would be increased problems from leaching of the soil, of the silt, and of plumbing systems that are already in place. It is a worry.

Senator **Mitchell**. Dr. Landrigan, in response to a question by Senator Chafee, during one of your answers, you said that it is a recognized scientific principle that studies which produce positive results should be given greater weight than studies which produce negative results.

Would you describe for me please what area of science within which that is recognized, and elaborate somewhat? To the layman it sounds like something that I agree with should be given greater weight than something that I disagree with.

Dr. **Landrigan**. Not if you are on the negative side of the issue. I think that maybe the best way to put it is to couch it in terms of Scottish jurisprudence. The Scots have three possible verdicts in a criminal trial. A person can be innocent, a person can be guilty, or there can be a third verdict which is "not proven."

I think the point is that many scientific studies, particularly epidemiologic studies, are inconclusive. Because epidemiology is hard to do, because people are exposed to many competing factors, because the ratio of signal to noise is low, if meticulous attention isn't paid to every possible confounding factor, it is quite conceivable in an epidemiological study that there could have been a signal which the investigator overlooked.

Therefore, a number of epidemiologic studies which are characterized as negative ought to be more properly to be characterized as not proven. That is what I mean when I say that a positive result ought to be given more credence than an allegedly negative result.

There are some studies, of course, that are very well done and produce a really well established negative result. Those studies ought to be given close scrutiny. But for every one of those, there are probably three or four studies which really are just inconclusive and are oversold as negative.

Senator MITCHELL. Thank you.

Dr. Godar, there will be entered into the record a statement by an organization which opposes acid rain control legislation, directed specifically at the question of how far we should go to protect asthmatic, and specifically exercising asthmatics.

The question is, is a more stringent standard to protect exercising asthmatics unreasonable?

Dr. GODAR. No. Someone this morning, in discussing this point, alluded to the canary in the mines. The canary doesn't mine coal, but is a warning that very soon the humans are going to suffer. The asthmatics have somewhat more sensitive airways than we do, however, their reactivity is virtually in direct proportion to the nature of the agent.

They are not reacting in this case to things to which they are allergic, they are reacting to irritant chemicals. Accordingly, if they react as asymptomatic asthmatics, not requiring medication, otherwise perfectly healthy, they basically are that 40 percent of that 20 percent who have hyper-reactive airways and are, in fact, known to be asthmatic, or a mere 8 percent of the entire population. I believe that the figure is actually higher.

They make an excellent model. The point is that experiments can only go on so long and, therefore, acute experiments tend to select a subject who will give a response within a reasonable, practical, economic time frame, accordingly, they make an excellent subject for study.

This does not imply that other subjected to similar activity would not show similar effects, albeit less. Therefore, for definitive results in a short period like two minutes of exposure to low levels of sulfur oxide, the asthmatic happens to be a very good subject.

The people studied were not having symptoms, they were not requiring medication, they were simply identified as having been asthmatics. They make an excellent subject for study.

Senator MITCHELL. Thank you, Dr. Godar.

My time is up on this round. In fact, I think I went beyond my time with my last question, and I apologize to you, Senator Stafford.

Senator STAFFORD. Thank you, Mr. Chairman. I really had a brief observation, and then a final question.

I suspect that many of our colleagues in the Senate feel that acid deposition is a problem, but it is a problem for some other part of the country, and it doesn't really affect me.

I am sorry that our new Member from Florida has left because I think many in Florida used to think that Florida was escaping most of acid rain deposition. I was interested to note, in one of the major newspapers the other day, that BMW has ceased off-loading their automobiles at Jacksonville in that State because acid rain has been damaging the paint finish on the cars during the two weeks that they have sat on the docks before being moved out. That would indicate to me that we can welcome Florida to the club of States that are being dumped on with acid rain, as Vermont is. It is even becoming a problem in our State with the quality of maple syrup, we are afraid, and we would hate to see that happen.

Let me finally make this observation, and ask you to agree or disagree with it, if you do, and if you do, why.

As I listened to you all this morning, I got the impression that nationally the degree of health effects, obviously, depends on the concentration of pollutants that might fall on any particular part of the country. Am I correct in coming to that conclusion?

I see you are all nodding affirmatively, and so I will take your answer as yes.

Mr. Chairman, I thank you very much.

Senator **Mitchell**. Thank you, Mr. Chairman.

This concludes the hearing. Gentlemen, thank you very much for your testimony, it has been very informative. The hearing is closed.

[Whereupon, at 11:19 a.m., the subcommittee adjourned.]

[Statements submitted for the record follow:]

American Academy of Pediatrics

STATEMENT

BEFORE THE

SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION

OF THE

UNITED STATES SENATE

COMMITTEE ON THE ENVIRONMENT AND PUBLIC WORKS

ON

ACID RAIN AND CLEAN AIR

PRESENTED BY

Richard M. Narkewicz, M.D., F.A.A.P.

February 3, 1987

Office of Government Liaison
1331 Pennsylvania Avenue, N.W.
Suite 721 North
Washington, D.C. 20004-1703
202-662-7460 / 800-336-5475

Good morning, Mr. Chairman, members of the Committee. I am Richard Narkewicz, M.D., President-Elect of the American Academy of Pediatrics and a practicing pediatrician in Burlington, Vermont. It gives me great pleasure to speak with you today, on behalf of the Academy, which represents more than 30,000 pediatricians committed to improving the health and well being of infants, children, adolescents and young adults.

I am here to discuss the health effects of air pollution on our children, with particular emphasis on acid air pollution. I can assure you that ozone, sulfates and the oxides of nitrogen cause disease in children and aggravate preexisting respiratory conditions. This is quite significant considering the prevalence of pediatric pulmonary diseases. Pediatric pulmonary disease accounted for 21 percent of all hospitalizations of children under 15 years of age in the United States in 1983. In as many as 350,000 other hospitalizations below age 15, a pulmonary disease was the secondary diagnosis. Each year these diseases are involved in about 1 million hospitalizations in children under 15 years of age. The length of hospital stay for pulmonary disease is longer than the average for all hospitalizations of children. As a primary diagnosis alone, the pulmonary diseases accounted for 4.8 million days of care or 29 percent of the total hospital days in children under 15 years of age in 1983. Acute pulmonary conditions among children under 17 years of age during 1981 accounted for an estimated 277 million days of restricted activity, 158 million days of bed disability and 90 million days lost from school. In 1980 and 1981 they accounted for 4.5 million office visits in children under 15. Asthma and chronic bronchitis are two of the more chronic pulmonary diseases in children. Asthma has shown an increased incidence in Eastern urban areas during the past 30 years, and was particularly severe in the 1960's when air pollution was at its worst. Asthmas attacks can be stimulated by a variety of factors, especially environmental pollutants. Many of these factors, such as weather, allergies and infections are difficult to control and have been a part of the environment for many generations. The more recently introduced industrial factors, which can be controlled, have a greater impact on the public health and are more amenable to intervention.

Children are more vulnerable than adults to pollutants of any kind, including those of the ambient air. This vulnerability begins in fetal life when a pregnant woman exposed to pollutants also exposes the fetus she is carrying. The cellular immaturity of children and their ongoing growth processes put them at further risk. Children's airways are much narrower than an adult's. The diameter of the bronchus at the root of an adult bronchial tree averages about one inch; the same bronchus in a child is about an eighth of an inch. A minor irritation caused by acid air pollution, which would produce only a slight response in an adult, will result in a dangerous level of swelling in the lining of the narrow airway of a child. Although children's airways are small, their metabolic needs for oxygen per unit mass are great. They breathe more rapidly and move more of the pollutant past airway and lung surfaces than do adults. Thus, they inhale more pollutant per pound than does an adult. Length of exposure is longer with children than adults. If during their early years of life a child is at high exposure, their risk of long-term damage increases. Another factor which makes children more vulnerable than adults to air pollutants is their increased rate of respiratory infections. Because of their immunologic immaturity, children have more acute infections of the respiratory tract than adults. The interactions of the infectious process with irritating pollutants intensify the pathologic process. Furthermore, the repair process for damaged airways is less efficient than in adults because of the small caliber of the airways and the development of partial obstruction that may result.

Mr. Chairman, I see many of these children with asthma and "wheezy bronchitis" in my practice. I always consider the role of air pollutants in any child suffering from respiratory difficulty. Nowhere in medicine does the axiom "an ounce of prevention is worth a pound of cure" play a more dramatic role than in pediatric pulmonary disease. I spend the major part of my time teaching the child and parents to use preventive medicine by avoiding the aggravating cause of asthma and bronchitis. Once the wheezing and gasping have started, it is difficult to control and usually leads to one or many office visits, often hospitalization and sometimes death. There is clear evidence that underlying hyper-reactivity (of the small bronchial tubes of asthmatics and children with bronchitis) is exacerbated by pollutants such as the oxides of nitrogen, sulfates and ozone.

In our roles as practicing pediatricians, we are frustrated because while we know some of the causes of respiratory difficulties, we have yet been unable to eliminate them from the environment. Thus we are forced to give our patients only symptomatic advice such as the following:

- stay indoors at times of severe pollution
- keep rooms as cool as possible
- use recirculated air if possible
- ban smoking in the home
- drink plenty of fluids.

Pediatricians have a duty to educate the public about hazards to the health of children. I would like to reiterate to this Committee that the ingredients of acid rain have been shown to adversely affect the respiratory tract and consequently the quality of health for children. They contribute greatly to morbidity and mortality rates. The sources of the pollution are being identified. Technology to significantly reduce pollutants has been in operation for many years in Japan. Doesn't it make more sense to eliminate the cause of the health problem, whenever possible, rather than having children suffer?

The Academy is grateful for this opportunity to work with your Committee in efforts to identify, reduce or eliminate the major sources of acid-air pollution. We recommend that strict measures be enacted to control emissions of sulfates, ozone and oxides of nitrogen from both industrial and automotive sources. If this goal is achieved, the health benefits will be especially enjoyed by children of this nation, who depend upon you for their public representation.

Thank you.

Office of Government Relations
Fran Du Melle, Director
1101 Vermont Avenue N.W. · Suite 402
Washington, D.C. 20005-3521
(202) 289-5057

AMERICAN LUNG ASSOCIATION
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STATEMENT OF THOMAS GODAR, M.D.

ON BEHALF OF

THE AMERICAN LUNG ASSOCIATION

TO THE

SUBCOMMITTEE ON ENVIRONMENTAL POLLUTION
COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS
UNITED STATES SENATE

RE: HEALTH EFFECTS OF ACID RAIN PRECURSOR POLLUTANTS

FEBRUARY 3, 1987

Mr. Chairman, members of the Committee, I am Dr. Thomas Godar, Director of the Pulmonary Disease Section at St. Francis Hospital and Medical Center, Hartford, Connecticut; Associate Professor of Medicine, University of Connecticut School of Medicine and Yale University School of Medicine; and in the active practice of chest medicine.

I am presenting comments today on behalf of the American Lung Association. In 1980, the Board of Directors of the American Lung Association developed its policy on the reauthorization of the Clean Air Act. Our principal concern then, as now, is the strengthening of the provisions of the Act to better control air pollution and thus protect public health. I and my colleagues in the American Lung Association and its medical section, the American Thoracic Society, have testified on numerous occasions before this Committee on the subject of air pollution health effects. We have provided the Committee with information demonstrating clearly the contribution of air pollution to the development or exacerbation of various lung diseases. The American Lung Association continues to place the highest priority on preserving and strengthening the federal government's program to control air pollution.

I appear before you today again with the very strong message that air pollution can and does have adverse impact on health--in some cases this impact affects large populations. In 1981 the ALA encouraged the Committee to consider the need to strengthen the health protection measures of the Act--today my message is the same but with greater urgency. The striking advances in information concerning health effects in the last several years justify this urgency.

I would like to summarize these advances and strongly encourage the Committee to hold additional hearings to review the extensive record of research findings that in my opinion should be the driving force for rapid reauthorization of the Clean Air Act. The research findings cited below are all clinical studies done at levels of air pollution commonly found in many regions of the United States today:

- the finding that healthy asthmatic subjects exposed to sulfur dioxide levels as low as 0.25 ppm for exposure periods as brief as 5 minutes when performing mild to moderate exercise can develop symptomatic and objective evidence of bronchoconstriction.

- the finding that exposure to levels of sulfur dioxide found in the ambient air combined with exposure to cold dry air potentiate the onset of symptoms in as little as 2 minutes. These effects are of particular concern in areas such as the Ohio River Valley where such levels of sulfur dioxide and severe cold weather are common in the winter months.

- the finding that short term exposures to 0.3 ppm of nitrogen dioxide potentiates exercise-induced bronchospasm and airways hyper-reactivity in healthy asthmatic subjects. Cold air is also found to potentiate this effect.

- the finding that, when combined with intermittent heavy exercise, exposure to ozone levels as low as 0.12 ppm can cause symptoms of airways irritation and decreased lung function in healthy subjects. These effects are reproducible in the same subjects when re-exposed several weeks to one year later.

- the finding that exposure to 0.12 ppm ozone in healthy subjects for a period typical of the normal workday--7 hours--for persons employed in outdoor jobs causes very significant decrements in lung function averaging reductions of 0.5 liter in FEV1.

Legislative proposals providing for reductions in total emissions of sulfur compounds as well as emissions of other pollutants are likely to have substantial public health benefits. Reduction in source emission rates will reduce the magnitude and frequency of short-term sulfur dioxide exposures. Also, by reducing sulfur dioxide emissions, concentrations of sulfate particulate matter will be reduced, resulting in fewer morbidity effects. However, to sufficiently protect public health, including the sensitive population of asthmatics, current findings document the need to limit exposures to less than 0.4 ppm of sulfur dioxide for durations of no more than 5-10 minutes. Translation of this into a short-term, one hour, standard including provision for an adequate margin of safety, would require a standard no higher than 0.20 ppm. To provide definitive health protection, the National Ambient Air Quality Standard (NAAQS) for Sulfur Dioxide must be strengthened by the addition of a short-term standard to ensure protection against adverse health effects at all locations in the United States. The NAAQS provides the primary mechanism for guaranteeing health protection. ALA firmly believes that the NAAQS program is an essential component of any program designed to protect public health, including sensitive populations.

I would like to conclude my comments by drawing the Committee's attention to another critical air pollution problem--nonattainment of the ozone NAAQS. As acid rain control legislation is pursued, the significance of the ozone nonattainment problem should no longer be ignored. ALA encourages the Committee to expand its legislative scope of concern. The research findings compiled as a part of the review of the ozone NAAQS indicate that the severity of the ozone problem is greater than many have thought. For example, EPA states that between 32 and 45 areas of the country will not attain the current ozone standard by the December 31, 1987 deadline. Further, current evidence suggests that to protect public health, the ozone standard should be strengthened--indicating that additional areas of the country may enter nonattainment status. Over 115 million people reside in current nonattainment areas, with approximately 20 percent considered to be sensitive as defined in the NAAQS provisions. This number could increase significantly if the ozone standard is not strengthened as the research findings suggest.

This is an important point, and I would like to review the range of populations considered sensitive to air pollution health effects. The determination of the 20 percent figure is estimated based on inclusion of the following groups:

- the pediatric population less than 2 years of age
- pregnant women
- patients with asthma, chronic bronchitis, and emphysema
- patients with a history of myocardial infarction, angina pectoris, and other forms of coronary heart disease
- persons 65 years of age and older.

EPA has developed a strategy to address the ozone nonattainment problem. EPA's approach however, fails to ensure that the continuing nonattainment problems will be resolved promptly and consistently across the country. This significant public health problem requires legislative remedy. The ozone nonattainment problem goes beyond speculation about the legality of the EPA initiative or the statutory significance and impact of the December 31, 1987 deadline for ozone attainment. This Committee must take leadership to improve and strengthen the Clean Air Act provisions for nonattainment areas to resolve the problems of long-term ozone nonattainment as expediously as practicable.

As the Committee continues its deliberations, ALA encourages you to consider action which leads to a comprehensive reauthorization of the Clean Air Act. ALA will continue to offer its scientific and medical expertise to assit you in assessing these complex health issues and appropriate changes in the Act for their resolution.

Thank you for this opportunity to comment.

Office of Government Relations
Fran Du Melle, Director
1101 Vermont Avenue N.W. · Suite 402
Washington, D.C. 20005-3521
(202) 289-5057

AMERICAN LUNG ASSOCIATION
The Christmas Seal People ®

March 12, 1987

The Honorable George Mitchell, Chairman
Subcommittee on Environmental Pollution
Senate Committee on Environment and Public Works
SD-410 Dirksen Senate Office Building
Washington, DC 20510

Dear Senator Mitchell:

Enclosed please find the answers to questions submitted to Dr. Thomas Godar as a result of the February 3 hearing on health effects of acid rain precursors.

If the American Lung Association can be of further assistance, please feel free to contact us.

Sincerely,

Fran

Fran Du Melle
Director

FDM:tsd

enclosure

SENATE COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS
SUBCOMMITTEE ON ENVIRONMENTAL POLLUTION

HEARING ON CLEAN AIR
THE HEALTH EFFECTS OF ACID RAIN PRECURSORS

FEBRUARY 3, 1987

RESPONSES TO ADDITIONAL QUESTIONS SUBMITTED FOR THE RECORD FROM SENATOR GEORGE J. MITCHELL CHAIRMAN, SUBCOMMITTEE ON ENVIRONMENTAL POLLUTION

1. Why should we protect exercising asthmatics? Do symptoms present in sensitive subpopulations such as asthmatics also signal health risks also present in the population at large?

Exercising asthmatics represent individuals with hyperreactive airways whose exposure to environmental irritants is intensified in the exercising state by virtue of the increased ventilation associated with exercise. 15-20% of the population has hyperreactive airways disease, with 5-8% having a diagnosis of asthma, representing a large segment of the population. In addition, this group represents a model of what might be happening to young children who exercise in the presence of environmental pollutants. The symptoms present in sensitive subpopulations such as asthmatics may signal health risks for the population at large.

2. Are the health effects which you have described limited to any particular areas of the country or are these effects observable throughout the country?

The health effects ascribed to the precursor pollutants of acid rain are in no way limited to any particular area of the country and are generally observable throughout the country where the National Ambient Air Quality Standards are not met. In the case of sulfur dioxide, the concern is about short term exposures for which there is no current standard.

3. Are the effects which you describe necessarily temporary or may some involve permanent damage?

Many of the health effects that are documented are done so by the use of models that permit a brief exposure to pollutants by sensitive individuals followed by a study looking for subtle changes in lung function or the presence of symptoms. These are only models, and although one cannot merely trnasfer the information obtained from models to the general population; the models permit the inexpensive and effective description of early changes and symptoms or pulmonary function defects that might otherwise require a massive population study over a long period of time.

The prolonged exposures followed in large population studies deomonstrate more permanent damage to lung function. Therefore, the effects described in experiments may frequently be temporary, while the effects described in large population studies may represent permanent damage to lung function and in the case of sulfur oxides represents a correlation between increased mortality and the concentrations of SO2 in the local air.

4. Are the health effects of acid rain precursors purely a medical problem? Is there an exclusively medical solution to these health risks? In the absence of new government regulation of air pollution, will the problem be cured through medical technology?

From the standpoint of the medical problems produced by **precursor pollutants of acid rain**, there is not at the present time acceptable medical solutions to these health risks, nor is it likely that such solutions will be developed in the near future. The problem of air pollution, therefore, will not be cured through medical technology but demands new government regulation to control air pollution.

5. Are there any studies which document the health effects of air pollution on neo-natal health? Are you able to draw any conclusions about the effect of air pollution on neo-natal health? If so please describe.

I am not in the field of neonatalogy and therefore can only comment regarding pulmonary related concerns. The primary concern in infants and young children is the incomplete development of the respiratory system and what effects air pollution may have on this sensitive population. The lung continues to develop until approximately age 8 and we know that any repeated exposures to irritants over time including air pollution, can cause significant decrements in lung function.

ADDITIONAL QUESTIONS FOR DR. GODAR

1. You state on page 3 of your testimony that "healthy asthmatics" develop bronchoconstriction when exposed to low levels of SO2 for 5 minutes while exercising. What is a "healthy asthmatic"?

With reference to the definition of a "healthy asthmatic", this represents an individual who is known to have bronchial asthma but in whom the asthmatic episodes occur either only with specific exposure to particular allergens or may occur as mild symptoms in the presence of a viral infection. Most of the time such individuals appear perfectly normal and function very much as you and I do, frequently able to partake in vigorous sports activity and appearing no different from the average citizen. Many such individuals have a history of asthmatic episodes but do not require medication on a regular basis.

2. Why wouldn't we expect to see some bronchoconstriction in exercising asthmatics apart from SO2 exposure?

A small segment of asthmatics do develop bronchoconstriction with exercise but generally when exposed to cold air during the exercise period. Patients are able to take medications to reduce the reactivity of the bronchial system, and in many cases may pursue their exercise or sports activity with minimal impairment. The exercise level described in the studies of SO2 exposure was generally well below the exercise levels that will induce bronchoconstriction in those asthmatics who have that experience with exercise.

3. You suggest in your testimony that approximately 23 million people (20% of 115 million) in nonattainment areas are "sensitive". What should the standard for ozone be to protect these populations?

Because effects have been demonstrated in clinical studies at the current National Ambient Air Quality Standard for Ozone--0.12 parts per million--it can be argued that such a standard does not provide a margin of safety as provided for in the Clean Air Act. If we are to protect the entire population including the sensitive individual, with such a margin of safety, the standard would have to be set below 0.12 parts per million.

The American Lung Association continues to support the standard-setting mechanism provided for in the Clean Air Act. Rather than speculating on where the standard for ozone might be set as a result of the current review being conducted by the Environmental Protection Agency, ALA encourages the completion of this process. This Committee may want to, as part of its hearings on the health effects of air pollution, review the standard-setting process.

ADDITIONAL QUESTIONS FROM MR. BAUCUS

1. The testimony presented at this hearing encompassed a wide range of children, from the unborn fetus to teenagers of 17 years of age. Is any specific age group affected more adversely than another?

The age group most effected by air pollution exposure is the age group from the new born to age two. The effects may thereafter dimish as the completion of the growth of the respiratory system is achieved at approximately age 8 years. Considerably more research is required to evaluate effects on the actively growing and physically active child.

2. Please describe the medical picture of a child suffering from chronic bronchitis or asthma?

The child suffering from chronic bronchitis or asthma would have cough, sputum prodution, and wheezing associated with tightness in the chest. Such a patient would experience difficulty in exposures to cold air, difficulty with exposures to dusts or virtually any irritant including the diesel exhaust from school buses, traffic, and exposure to second-hand smoke. This patient may spend a number of evenings in the emergency room receiving intravenous medication and other support services for acute episodes. There may be limits in sports activity in may cases. Patients often require medication on a daily basis and may have to have a limited exercise program with special physical activity different from their peers, might spend a number of days home with acute episodes, away from class and, therefore, have more academic problems in maintaining grades. In addition, such individuals would run an increased risk of acute episodes when exposed to the ordinary winter months' viral infections that are so prevalent in the schools.

3. Are we seeing any changes in asthma and bronchitis attacks throughout the country?

There appears to be an increased incidence of asthmatic attacks in the personal experience of clinicians such as myself but the study of these trends include so many factors including improvement in our identification of bronchitis and asthma that it is difficult to know if increasing numbers represent improved diagnostic facilities and acumen, special situations during given winter months secondary to prevalent viruses, or other factors such as ambient air pollution.

4. As noted in written testimony, 21% of all hospitalizations of children under 15 years of age in the United States in 1980 was due to pulmonary disease. How do these figures vary for different states or regions of the country? How do they vary between urban and rural areas? What about a state like Montana?

As noted in my testimony I practice adult pulmonary medicine. As these statistics were not in my testimony I do not know the source and therefore cannot speculate about regional or deomographic differences.

5. Testimony also noted that 4.8 milliion days of care, or 29% of total hospital days for children under the age of 15, is for pulmonary disease. What is the cost of this care?

Again, since I did not include such information in my testimony and am unfamiliar with the source of this information, I cannot answer this question accurately.

6. In your opinion, how would the lives of children suffering from respiratory distress be changed by reducing the ambient levels of acid rain precursors? How would this translate in terms of a reduction in the number of hospitalizations for pediatric pulmonary disease?

I believe children who have respiratory problems and experience exposure to **precursor pollutants of acid rain** would have less problem with acute episodes and would have better control of their respiratory condition translating into a reduction in susceptibility to exacerbations during vial infections with a reduction in hospitalizations for pediatric pulmonary disease. Past studies documenting an increased incidence of emergency room visits, treatments for pulmonary infections, or hospitalizations in urban industrialized communities compared to rural areas would give one every reason to believe a significant reduction in health care costs and in risk to the pediatric population would occur with reductions in emisssions of these pollutants.

7. What type of long-term damage is being caused in children breathing air pollutants?

The so-called Harvard Six-Cities Study has found that air pollution, particularly fine particles associated with total sulfate particulates, produces respiratory symptoms in school children. In some instances marked decrements in lung function have been noted. This study, in part, is attempting to identify long term effects by following a group of children in clean cities and in so-called dirty cities over time.

8. Does air pollution cause a child's immune system to be less effective in warding off disease, or does a less developed immune system allow for greater incidence of air pollution-induced infections?

It is not clear at the present time if a child's immune system is less effective than the adult system in warding off disease as a consequence of air pollution or if that is, in fact, only the natural state of the young child. Certainly the immune system of a small child is poorly developed in the absence of air pollution, and therefore, it would be my "educated guess" that a less developed immune system would permit greater incidence of air pollution-induced infections as a consequence of their natural incomplete immune system development.

9. Do we know enough about the public health problems caused by air pollution for the standards to be tightened?

Yes, there is certainly data sufficient to suggest that a short-term standard for SO2 is necessary and that our current SO2 standard should not be weakened. In addition, there is evidence that ozone has deleterious health effects at levels at or below the current standard of 0.12 parts per million, suggesting that the standard should be strengthened to adequately take into account the established evidence of health effects.

10. The Clean Air Act requires EPA to review air standards every three years. Is this review occurring? If it were, would EPA be compelled to tighten the standards?

EPA continues to review the standards as required by the Clean Air Act. This review is exceedingly complex due to many factors including the types of scientific research reviewed, the often rapid growth and change in the body of scientific evidence, the range of interests participating in the process--affected industries, environmental and health groups, and the very limited staff and budget resources allocated by the Agency to this review process.

As to whether such review should compel the Agency to tighten the standard, the ALA is concerned that often the decision is politicized by involvement of the Office of Management and Budget rather than based on the scientific process envisioned by the authors of the Clean Air Act and originally used to set standards.

11. EPA is either unwilling or unable to set a standard that protects public health. Should Congress statutorily set this standard?

ALA believes that Congress should explore mechanisms through the Clean Air Act reauthorization to address improving the standard-setting process including resource allocation for the process. Defining the process and providing oversight for the process are more appropriate functions for Congress.

12. You indicated that there have been striking advances in our knowledge of the health effects of air pollution. Briefly, what are some of these findinggs? Has EPA reviewed these studies? Will you supply a detailed list of these studies to the Committee?

The striking advances in our knowledge of health effects of air pollution are the results of intensified research on health effects, largely in chamber studies and in acute exposure experiments under very controlled circumstances permitting us to see subtle changes at early stages not previously identified. Many of these studies have occured at EPA's clinical research facility on the campus of the University of North Carolina-Chapel Hill, or have been funded by the National Institutes of Health.

As noted in my testimony, among the striking findings are:

- o healthy asthamtic subjects exposed to sulfur dioxide levels as low as .25 parts per million for periods as brief as 5 minutes when performing mild to moderate exercise can develop symptomatic and objective evidence of bronchoconstriction
- o exposures to levels of sulfur dioxide found in the ambient air combined with exposure to cold dry air potentiate the onset of symptoms in as little as 2 minutes
- o short term exposures to 0.3 parts per million of nitrogen dioxide potentiates exercise-induced bronchospasm and airways hyperreativity in healthy asthmatic subjects
- o when combined with intermittent heavy exercise, exposure to ozone levels as low as 0.12 parts per million can cause symptoms of airways irriation and decreased lung function in healthy subjects.

Rather than duplicate the massive bibliographies prepared by the EPA as part of the preparation of criteria documents, I suggest that you request the Agency to supply you with the bibliographies from these documents.

13. A large portion of the testimony has been devoted to the adverse health effects of acid deposition on children. Would you briefly describe the impact of acid deposition on children. Would you briefly describe the impact of acid deposition on the health of the elderly? What is the cost of this care to Medicare?

Children are considered to be part of the senstive population becuase of their immature respiratory system. Further, because of the increased level of exercise and increased rates of ventilation, they may recieve higher exposures. Also of concern is the child with a pre-existing respiratory condition such as asthma. The **precursor pollutants of acid rain** contribute to the general air pollution problem thus impacting the health of this subpopulation.

The elderly on the other hand are at risk and included in the sensitive population because of a decline in the pulmonary defense mechanisms associated with aging. This leads to a decreased ability to cope with air pollutants and other respiratory irritants. Again as with children, the elderly with pre-existing respiratory problems are especially at risk since air pollution can cause exacerbation of conditions such as Chronic Obstructive Pulmonary Disease.

The medical costs of these exposures and the increased incidence of infection and respiratory distress is difficult to document. Certainly there is no current methodolgy to assess the portion of Medicare expenditures attributable to air pollution exposures.

QUESTIONS SUBMITTED BY SENATOR LAUTENBERG
1. Do you feel that current medical and scientific knowledge justifies Congress taking action now to control sulfates, nitrogen oxides, and ozone?

Yes, I believe medical and scientific knowldege is adequate to justify further action. Although further research should continue, the need for information does not remove us from responsibility for controlling health effects that are currently recognized.

2. You have commented on the health effects caused by the pollutants associated with acid rain. You have limited your comments, I assume, to the effects caused by current air quality levels.

The comments on health effects caused by **precursor pollutants of acid rain** have been limited to problems produced by current air quality levels. I believe that if further controls on emissions of these pollutants are not adopted levies will increase in many areas.

3. You have described health effects of pollutants realted to acid rain. I would like to get a concrete, layperson's sense of what kind of health effects we are discussing here.

Sulfur dioxide and sulfates irritate the respiratory system and are responsible for reduced pulmonary function. Of particular concern is the sensitive population of individuals with hyperreactive airways--15-20% of the population with 5-8% of this population diagnosed with asthma. We have found that this group of sensitive individuals is at higher risk when exposed to sulfur dioxide. Risk includes development of symptoms of objective bronchoconstriction. This could lead to an asthma attack if the exposure is continued or the concentration of the exposure is increased.

Nitrogen dioxide is a respiratory tract irritant and those with respiratory conditions are particularly at risk of effects when exposed to this pollutant. It is also responsible for reduced pulmonary function and reduced resistance to infection.

Oxidants are another pulmonary irritant and exposures at levels of the current standard can produce significant decrements in lung function. This pollutant is also being studied for its role in pulmonary edema. Recent research demonstrates that healthy exercising adults react to exposure levels at or near the current standard.

The number of individuals in the country at risk of experiencing harmful effects is estimated at 20%. This includes infants and young children, the elderly and individuals with pre-existing conditions such as asthma, Chronic Obstructive Pulmonary Disease and chronic bronchitis.

4. Do you think the federal government is making sufficient commitment to research on the causes and effects of acid rain?

The federal government has not done enough in terms of research related to the health effects of air pollution in general or in development of control technologies. Although there should be major funding increases for this type of research, action by EPA and Congress does not require results of further research since the case is well made for stringent controls, especially over the **precursor pollutants of acid rain**.

There should be further stimulation of research in health effects of air pollution, an area which suffers from inadequate resources. This would attract new talent into the area of air pollution health effects research. Further, there is not adequate coordination of research efforts by the different federal agencies and departments, and in general, not enough research has been carried out given the pervasiveness of the problem of air pollution.

As a further example of the lack of resources, the President's proposed budget for FY 88 would continue the cuts in the EPA Air Program, now totalling 24% since 1981. It is difficult to carry out any regulatory mandate let alone carry out that mandate well with these sorts of cuts.

5. Dr. Godar, a problem which you emphasized is ozone nonattainment. This is a significant problem for New Jersey, which is not expected to meet the 1987 deadline.

Ozone nonattainment is indeed a very significant problem for New Jersey. Ozone produces exacerbation of existing airway disease, particularly during the summer months when there is sufficient sunlight to alter the precursors and generate ozone levels notorioulsy well above the standard. In addition, the ozone generated in the New Jersey/New York areas drifts northeast to Connecticut, Massachusetts, and Rhode Island providing a wide band of nonattainment during the summer months.

There are specific health effects of ozone including the exacerbation of asthma, increased shortness of breath, cough, and respiratory distress in patients with Chronic Obstructive Pulmonary Disease. There are other effects such as sore throat, headache, and nasal irritation. My personal experience with patients who have emphysema, bronchitis, and asthma who leave high ozone levels during the summer months to vacation where the air is cleaner leads me to believe that the symptoms correlate very well with ozone levels since some patients have a recurrence of symptoms within hours of returning to their homes in areas where the ozone standard is exceeded.

6. Dr. Godar, you mention in your written testimony on pages 3 and 4, health problems associated with ozone levels of .12 parts per million.

Yes, there are several studies which document effects at .12 parts per million ozone, the current standard. Because we are seeing effects at this level, it can be argued that such a standard does not provide a margin of safety as provided for in the Clean Air Act. If we are to protect the entire population, including the senstive populations, a standard for ozone would have to be below .12 parts per million.

7. Dr. Godar, do you think EPA's ozone strategy is insufficient?

Yes, I believe EPA's ozone strategy is insufficient. Inadequate progress toward attainment by many states has not had adequate attention by EPA. I believe appropriately applied sanctions are required to produce meaningful progress; for example, in those states that have failed to implement an effective inspection and maintenance program, a requirement for an extension of the deadline to 1987.

8. Dr. Godar, you indicated in your written testimony on page three that cold, dry weather can worsen some of the health effects associated with sulfur dioxide and nitrogen dioxide.

Cold, dry air will increase the health effects associated with sulfur dioxide

-nd nitrogen dioxide. This has been demonstrated in several clinical studies using healthy exercising asthmatic subjects. This is a particular problem in areas of the country such as the northeast which experience high levels of this pollution during winter months.

SENATE SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION

HEALTH EFFECTS OF ACID RAIN

STATEMENT OF

BAILUS WALKER, JR., Ph.D., M.P.H.
COMMISSIONER OF PUBLIC HEALTH
COMMONWEALTH OF MASSACHUSETTS

ON BEHALF OF THE

AMERICAN PUBLIC HEALTH ASSOCIATION
1015 15 STREET, NW
WASHINGTON, DC 20005
202-789-5600

FEBRUARY 3, 1987

Good morning. I am Dr. Bailus Walker, Jr., President-Elect of the American Public Health Association (APHA). APHA, with more than 55,000 members, is the world's oldest and largest organization of public health professionals. We are pleased to have the opportunity to testify before you today.

Acid rain is the quintessential modern environmental health issue.

Its effects are related to events that are initiated through a variety of complex processes and occur at times and places that may be far removed from the actual source of the pollution.

More often than not, the sources of acid rain and the receptors are in different political jurisdictions, often not even contiguous ones, which raises both scientific and political questions which this Subcommittee needs to address.

Much public attention has focused on the impacts of acid rain and its components, sulfur dioxide (SO_2) and nitrogen oxides (NO_x), on the environment. Aquatic ecosystem disruption and damage have been well documented. Growth declines in forests have been noted both here and in Europe. Historic buildings and other external building materials have been damaged.

But we are concerned that in all the debate on acid rain legislation, the health effects from exposure to acid rain have almost been treated as secondary effects. As a public health organization, we come before you today to say that acid rain affects more than just fish or trees --- it directly and indirectly affects the health of people.

The public health community is encouraged by the evidence of air quality improvements in the United States.

In the overall effort to reduce air pollution, the U.S. is ahead of its neighbors in at least one area - automobile emissions.

But improvement has not been uniform and some new patterns of air pollution are emerging.

Moreover, the fact that ambient air quality has improved since 1970 by traditional measurements and national standards, unfortunately, does not necessarily imply that human exposures to those pollutants of public health concern have decreased.

Nor does it imply that the population at risk - infants, children, the elderly - has decreased.

Indeed, it is clear even to the most casual observer that evidence from many sources is sufficient to warrant concern that current air pollution, including acid rain, produces substantial health effects in some segments of the U.S. population.

These effects include a variety of acute and chronic respiratory problems that have been linked with previous kinds and amounts of pollution.

Acute respiratory effects of concern include an increase in the frequency of respiratory asthmatic attacks, deficits in pulmonary function, and increases in the frequency of respiratory diseases in both children and adults.

Although much attention has been focused on respiratory disease end points, air pollution may be a contributing factor to other major public health problems, such as neurobehavioral effects in children and hypertension.

It is plausible that air pollution causes some part of the lung cancer incidence in the United States but the size of the fraction is not known. Moreover, children and elderly people, together with sensitive populations such as those individuals with respiratory diseases, are particularly susceptible to the impacts of air pollution.

Unfortunately, without effective and aggressive federal action, it appears that the adverse health effects from acid rain will only increase. Sulfur, a precursor to acid rain, is a pervasive pollutant in the environment. On an annual basis, it has been estimated that more than 25 million tons of SO_2 are emitted (National Acid Precipitation Assessment Program, 1983).

Electric utilities, industrial boilers, smelters and refineries are major sources of SO_2. While environmental controls on some of these facilities have reduced emissions at certain plants, there is now concern that total SO_2 emissions may actually increase with the use of tall stacks and from the burning of high sulfur coals (National Clean Air Coalition, 1984).

Summarized below are documented direct and indirect health effects resulting from exposure to acid rain.

DIRECT HEALTH EFFECTS

Historically, there has been much data on health effects from exposure to sulfur dioxide. Acute air pollution episodes going back to the 1950s in London and to 1948 in Donora, Pennsylvania have provided us with information on air pollution and its effects on human health. In the time since those episodes, studies have shown that harmful health effects have been occurring at lower and lower levels of air pollution (Jay, 1986). Moreover, even short-term exposures to sulfur dioxide have been shown to have effects on pulmonary function.

Clinical studies using human subjects have indicated that both healthy and at-risk persons are vunerable to the effects of sulfur dioxide. The actual magnitude of the response varies, and is dependent on such factors as health status, individual sensitivity, and activity level (Jay, 1986).

Studies of normal adults at rest have indicated that bronchoconstriction occurs at sulfur dioxide concentrations of 1.0 to 2.5 parts per million (ppm) (Lawther, 1975; Melville, 1970). In one study, where the subjects were exposed to SO_2 pollution by mouth inhalation only, bronchoconstriction was reported to occur at concentrations of 1.0 ppm (Amdur et. al., 1953), indicating that breathing through the mouth increases SO_2 penetration (Melville, 1970). This is of particular interest since about 15 percent of the general population are mouth breathing (Commonwealth of Massachusetts, 1984).

While the health effects on healthy adults are quite clear, the effects are even more pronounced in individuals with hyper-reactive airways, including asthmatics and those with allergies (Jay, 1984). In asthmatics, researchers have observed bronchoconstriction, sometimes accompanied by shortness of breath and wheezing, at SO_2 levels less than 0.5 ppm (Kagawa, 1979; Sheppard, 1981). Studies have shown that asthmatics can be up to 20 times more sensitive to the effects of sulfur dioxide than healthy adults (Koenig, 1980, 1981, 1982).

The health impacts from exposure to sulfur dioxide to young and old populations have been well documented. Studies on the impacts to children of high acid deposition have been reported by the Canadian government (Stern, 1984). In a 1983 study, children who lived in the high acid deposition community of Tillsonburg, Canada exhibited a small (2%) but statistically significant decrease in lung function, resulting in reduced lung capacity to process air as well as a reduced ability to expel inspired air (Stern, 1984). These children also had a greater prevalence of respiratory symptoms, including persistant chest colds, allergies, and coughs (Stern, 1984).

It is estimated that there are about 62 million children under 17 years of age, of which 46 million are under 14 (Jay, 1986). Because the lung and immune system of children are still developing, they provide incomplete protection from sulfur dioxide and other pollutants. Also, because children spend more time outdoors and tend to be engaged in more active activities than adults, they tend to experience greater exposure to pollutants from acid rain because of higher ventilation rates (Calabrese, 1978; Becklake, 1978).

Another sensitive population is that of senior citizens. There are approximately 24 million elderly people 65 years or older (Jay, 1986). Besides immunological deficiencies, many senior citizens have lost some lung function, making them more vulnerable to elevated levels of pollutants (Eisen, 1976; Cotes, 1979).

INDIRECT HEALTH EFFECTS

Besides the direct health effects listed above, there are indirect health effects resulting from contact with media contaminated by SO_2, such as drinking water.

In a 1982 study, the U.S. Environmental Protection Agency found that acid precipitation had significantly increased the acidity of both surface and shallow groundwater in many parts of the country. In my own state of Massachusetts, it is estimated that 1,000 of the more than 2,600 lakes, ponds, and reservoirs are sensitive to acidification (Commonwealth of Massachusetts, 1984). Twenty percent of these, including the Quabbin Reservoir which supplies water to the City of Boston, are on the verge of losing their capacity to neutralize acids and between 5 and 10 percent of these are already acidified.

Indeed, an EPA study released just three weeks ago indicates that western surface waters remain relatively unspoiled by acid precipitation, while in the east, more than 10% of the lakes in the Adirondack mountains of New York are on the verge of being acidic.

The increase in acidity of drinking water can have serious public health impacts due to its effects on the solubility of metals (Commonwealth of Massachusetts, 1984). This can be a serious problem in many older urban centers, where antiquated water systems consist of lead and copper pipes. Acidic water can leach these metals out of the pipes into the water supply. Besides lead and copper, aluminum and mercury can also be leached.

The health effects from exposure to lead are well known: headaches, dizziness, anemia, brain damage, general mental impairment, and eventually, possible death.

Lead has also long been recognized as toxic to the kidney. Prolonged high-dose exposure to lead both in children and in adults has been shown to produce kidney dysfunction which can lead to secondary hypertension.

Children are also exposed to lead from ingestion (as in lead paint chips) or through inhalation. Lead poisoning occurs when stored body levels of lead gradually accumulate as intake exceeds excretion. Thus, with incremental amounts of lead from drinking water, such exposure can increase the lead body burden beyond the acceptable health threshold (Vengropungal and Kuckey, 1978).

Acidic water supplies have also become contaminated from copper pipes. While no definitive studies have yet been conducted on health effects from exposure to high copper levels, it has been observed that acidic foods or drinks that have prolonged contact with copper have caused acute copper toxicity in those who ingest it (Commonwealth of Massachusetts, 1984). The symptoms include vomiting, hypertension, coma, jaundice, and in extreme examples, possible death.

Acidic waters have also been shown to consistently have higher levels of aluminum than more basic water. While no aluminum standard currently exists, observed cases of aluminum poisoning have involved some of the following conditions: encephalopathy, osteomalacial (bone softening), and anemia (Wills and Savory, 1983).

Some biomedical scientists have also indicated that increased concentrations of aluminum may be associated with Alzheimer's Disease. Coyle, et. al. observed that brain tissue of Alzheimer's victims had elevated levels of aluminum (1983). However, the evidence that aluminum causes Alzheimer's Disease is largely circumstantial at present, and will no doubt require additional study (Commonwealth of Massachusetts, 1984).

In addition, acidic water increases the solubility of mercury as well as its availability for bioconcentration in the food chain. As a result, large predatory fish at the top of the food chain tend to have higher levels of mercury (Philip, et. al., 1980). Thus, individuals and cultures which eat large quantities of fresh-water fish from acidic water bodies are at greater risk of mercury poisoning.

Studies have indicated that in highly acidic waters, residues of mercury in fish tissue increases (Vernelov, 1980). This may be due to several factors: at a lower pH level, mercury is transformed to methyl mercury, which is then more readily absorbed (Tomlinson, et. al., 1979); in acidic waters, mercury is more water soluble, contributing to higher concentrations (Miller and Akag, 1979); and acidic waters affect the permeability of cell membrane, thus increasing mercury absorption (Maetz, 1971).

CONCLUSION

In conclusion, the adverse health consequences from exposure to acid rain and its precursors of both healthy and high risk individuals have been well documented. APHA calls upon Congress to move forward and begin to reduce acid rain in an effort to protect human health and welfare.

Given the current scientific knowledge about the phenomenon of acid deposition, it is easy to suggest that policymakers face a dilemma.

They do not.

The choice is clear. We must not risk further health damage by delaying effective control of acid rain until we have answers to every single scientific, technical, or economic question or until we have the perfect solutions.

We need not wait for more proof, beyond all reasonable doubt, that acid rain is harmful or has already damaged human health and welfare or resort to other strategies designed to cause continuous delay in remedial action.

We should take into full account the large body of information that deals with the sources of acid deposition and the seriousness of its effects, and move forward with aggressive federal action to interrupt current human exposures to acid deposition and prevent future environmental degradation and human health effects.

Acknowledgement

I thank Vivien Li, Special Assistant to the Massachusetts Commissioner of Public Health, for her assistance in the preparation of these remarks.

Testimony of

Philip J. Landrigan, M.D., M.Sc., F.A.A.P.
Professor of Community Medicine and Pediatrics
Director, Division of Environmental and Occupational Medicine
Mount Sinai School of Medicine
New York, New York 10029

Acid Air Pollution and the Health of Children

Before the
Committee on Environment and Public Works
United States Senate
Washington, D.C.

Tuesday,
February 3, 1987

ENVIRONMENTAL AND OCCUPATIONAL MEDICINE
DEPARTMENT OF COMMUNITY MEDICINE
MOUNT SINAI SCHOOL OF MEDICINE OF THE CITY UNIVERSITY OF NEW YORK
NEW YORK, NY 10029
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Philip J. Landrigan, M.D., M.Sc.

Mr. Chairman and Members of the Committee.

My name is Philip J. Landrigan, M.D., M.Sc. I am Professor of Community Medicine and Director of the Division of Environmental and Occupational Medicine at the Mount Sinai School of Medicine in New York City. I am a pediatrician and also a specialist in preventive medicine. I am Chairman of the Committee on Environmental Hazards of the American Academy of Pediatrics. In 1985, I was Co-Vice Chairman of the Committee on the Epidemiology of Air Pollutants sponsored by the National Research Council of the National Academy of Sciences. My curriculum vitae is attached. I am pleased to appear before you today to discuss the effects of acid air pollution on the health of children.

Current Status of Acid Air Pollution in the United States.

Ambient concentrations of many air pollutants, such as sulphur dioxide and particulates have decreased sharply over most of the United States since passage of the Clean Air Act in 1970. Lethal epidemics of acute respiratory illnesss caused by serious episodes of air pollution ("killer fogs") once were common. Today, they are no longer observed.

However, the current picture in air pollution is not one of uniform improvement. High local concentrations of air pollutants are seen in certain cities in relation to busy highways and industrial complexes. Patterns of pollutant dispersal have changed, partly as the result of recent control measures such as higher stacks and widespread installation of catalytic converters on automobiles. Sulfate concentrations, although lower than in the past, are still unacceptably high in many areas, particularly during the summer months. Finally, and perhaps most important to this morning's hearing, levels of two air pollutants, nitrogen oxides and ozone have not declined in recent years. Ozone concentrations have in fact shown some recent increases.

Health Effects of Acid Air Pollution.

Several lines of converging evidence - epidemiologic studies of populations, controlled studies of human volunteers, and toxicologic studies in cell systems and animals - all indicate that current levels of acid air pollution are able to produce substantial adverse health effects in certain segments of the American population and particularly in children. Today, I should like to review this evidence with you.

The Harvard-Six-City Study. The Harvard-Six-City study is a prospective epidemiologic study of persons in six municipalities across the United States: Steubenville, Ohio; St. Louis, Missouri; Watertown, Massachusetts; Kingston-Harriman, Tennessee; Topeka, Kansas; and Portage, Wisconsin. These cities represent a spectrum in severity of air pollution. Air pollution data in these cities are collected from outdoor and indoor monitors and are correlated with indicators of pulmonary function and lung disease in selected adults and children.

The Harvard-Six-City study has found that exposure to acid air pollution, particularly to fine particulates associated with total sulfate particulates, produces respiratory symptoms in school children. A marked increase in prevalence of bronchitis occurs as the concentration of fine airborne particles increases from 12 to 37 micrograms per cubic meter ($\mu g/m^3$). This effect is seen both in children with a prior history of wheezing, as well as in children without such history. However, the effect is greater among children who wheeze.

Canadian Studies. In a series of studies conducted in Southwestern Ontario between 1974 and 1978, associations have been sought between airborne concentrations of particulates, sulfates, oxides of nitrogen and ozone and the frequency of hospital admission in children. In the winter months (January and February), a weak correlation was observed between airborne levels of sulfates and excess total hospital admissions. In the summer months (July and August), excess admissions to hospital for respiratory conditions were significantly correlated with levels of sulfate and ozone, as well as with temperature. Additionally, admissions due to asthma were found to be strongly correlated with ozone levels. No significant relationship was found between air pollution and admission to hospital for any cause except respiratory illness.

More recent Canadian studies, conducted in the summers of 1983 and 1986, have examined pulmonary function among children in summer camps. These studies have sought to relate changes in pulmonary function to changes in concentrations of atmospheric sulfates, oxides of nitrogen, ozone, sulfuric acid, and particulates. These studies indicate that children exhibit transient decreases in lung function in association with elevated levels of air pollutants. Further, it is important to note that these effects were evident both in asthmatic children as well as in children with no prior history of asthma. The effects are therefore manifest across the entire population.

New Jersey-Pennsylvania Studies. Studies of children in summer camps have also been undertaken in New Jersey and Pennsylvania by investigators from New York University. The study design was similar to that employed in the Canadian studies. These analyses also found that increased exposures to ozone are associated with consistent decreases in pulmonary function, particularly in indices of expiratory lung function, in young children at summer camp.

The most recent of the New Jersey-Pennsylvania studies, conducted in the summer of 1982, had the opportunity to monitor respiratory function in children during and after a large-scale regional episode of photochemical smog. That episode produced high concentrations of particulate matter, including sulfates, as well as ozone. The data indicate that peak expiratory flow volume was decreased in children during that episode. Further, this depression in pulmonary function persisted for as long as a week after the end of the smog period before finally returning to normal.

Summary of Epidemiologic Data. In summary, the epidemiologic data from these studies indicate quite clearly that current atmospheric levels of ozone, sulfate, and particulates are capable of producing transient decrements in pulmonary function of children. These decrements in function may last longer than the episodes of pollution themselves and can persist for at least a week after cessation of the pollution episodes. These decrements in function appear to be associated with increased frequency of asthma and, at least during the summer months, with increased frequencies of hospital admission.

Full characterization of these phenomena is not yet available. Properly conducted epidemiologic studies of the acute and subacute effects of ambient air pollution on children are few in number. However, these studies do indicate clearly that current levels of acid air pollution have adverse effects on children's health. Further, they indicate that these effects are sericus. The full significance of these effects and their possible long-term consequences await further study.

Controlled Studies of Human Volunteers. Controlled studies of human exposures to mixtures of aerosols that simulate actual environmental exposures provide an opportunity to corroborate the results of epidemiologic studies.

Studies of asthmatic subjects exposed to acid aerosols at the University of Rochester have shown that sulfuric acid mists produce pulmonary symptoms and effects on lung function at levels of exposure below the levels required to produce similar effects in normal persons. Furthermore, sulfuric acid aerosols were found to produce symptoms of increased airway reactivity in normal subjects after a lag period of 24 hours. These studies suggest that all persons in the population are susceptible to respiratory dysfunction following exposure to sulfuric acid, but that asthmatics are particularly sensitive.

Studies conducted in Canada of human volunteers exposed to 0.75 ppm ozone for two hours in a chamber also demonstrated significant decreases in pulmonary function. Further, in subjects who exercised intermittently during ozone exposure both pulmonary symptoms as well as decreases in pulmonary function were markedly accentuated. The authors of that investigation concluded that this level of exposure to ozone, even in the absence of other pollutants, is too high for even brief exposure of the general population.

Recent studies at the University of Rochester have shown that healthy, asymptomatic non-smoking adult males exposed to low-levels of nitrogen oxides, insufficient to produce any pulmonary functional deficit have enzymatic changes in their lungs and bronchi. Furthermore, in the same laboratory, low-level exposure to nitrogen dioxide was found to produce progressive decrements in forced expiratory volume after exercise in subjects with chronic obstructive lung disease. These findings suggest that persons suffering from chronic lung disease might be a subgroup within the population particularly at risk for exposure to acid air pollutions including acid aerosols.

Animal Toxicologic Studies. Toxicologic studies in animals provide yet a further opportunity to corroborate the results of epidemiologic studies on the toxic effects of air pollution. A major study of the responses of experimental animals to air pollutants is currently underway at New York University. This study has shown that repeated exposures to sulfuric acid mist at modest exposure levels and for short daily exposure times resulted in production of hyperresponsive airways in previously healthy animals. These data are consistent with epidemiologic studies in the Ontario summer camps. Further, once the airways become hyperresponsive, subsequent exposure to other stimuli such as air pollutants or cold air have been shown to result in bronchoconstriction similar to that seen in asthma.

Conclusions:

All of these data confirm that exposure to current, relatively low levels of air pollution can produce toxic effects in the lung. These effects are not benign. Nor do they represent a form of adaptation of the respiratory tract to pollutant exposure. They are, in fact, pathologic effects. Whether they have long-term consequences remains to be seen. Additional research will be required to define whether there are delayed effects. Further research will be required also to define precise levels of which these adverse effects occur.

However, it seems reasonable at the present time to conclude that ambient exposures to ozone should at all times be kept below 100 ppm, and that ambient exposures to sulfates should at all times be kept below 20 $\mu g/m^3$. Full data to support those recommendations are not yet in hand. However, the available data are sufficient, and sufficiently alarming to justify the imposition now of strict standards without awaiting the results of further more detailed research. In the present circumstances, the regulatory agencies cannot afford to wait for more precise data on adverse effects. Good qualitative and semi-quantitative data are available now. Considerations of cost and benefit pale when applied to the health of children. The task confronting the regulatory agencies today is to exercise their mandate to protect the public health. To delay action pending completion of additional research and to take steps to protect children's health only after irrefutable evidence of disease and damage have accumulated is poor medicine and poor public policy.

Thank you. I shall be pleased to answer any questions.

PJL:ss
Att.

THE MOUNT SINAI MEDICAL CENTER
ONE GUSTAVE L. LEVY PLACE • NEW YORK, N.Y. 10029

Mount Sinai School of Medicine • The Mount Sinai Hospital

Philip J. Landrigan, M.D., M.Sc.
Director, Division of Environmental
and Occupational Medicine
Department of Community Medicine

(212) 650-6173

March 11, 1987

The Honorable George J. Mitchell, Chairman
and
The Honorable John H. Chafee
Committee on Environmental and Public Works
United State Senate
Washington, DC 20510-6175

Dear Senators Mitchell and Chafee:

Thank you again for having invited me to appear before the Subcommittee on Environmental Protection on February 3. Thank you also for having sent me the long and thoughtful lists of supplementary questions. I welcome the opportunity to elaborate further on the issues which we discussed at the hearing.

Additional Questions from Senator George J. Mitchell.

1. Why should we protect exercising asthmatics? Do symptoms present in sensitive subpopulations such as asthmatics also signal health risks also present in the population at large?

   Data from summer camp studies conducted in Canada and the United States indicate that all children, both asthmatics and non-asthmatics, exhibited reductions in pulmonary function during episodes of acid air pollution. However, these changes were magnified in asthmatic children, especially in exercising asthmatics. Thus, asthmatic children constitute a particularly vulnerable subset of the population. These children are deserving of special protection under the Clean Air Act, but additionally it should be recognized that they signal health risks for the population at large.

2. Are the health effects which you have described limited to any particular areas of the country or are these effects observable throughout the country?

   The health risks which I described may be found in any area of the country in which acid air pollution is present. The Harvard Six Cities Study indicates that the worst pollution in the six municipalities examined was to be found in Steubenville, Ohio and St. Louis, Missouri.

3. Are the effects which you describe necessarily temporary or may some involve permanent damage?

Sufficient long-term data are not yet available to determine with certainty whether the effects I described involve permanent damage to the lungs.

However, it is significant to note that the decreases in pulmonary function which were observed to occur in children following exposure to acid aerosols persisted for as long as four to five days after dissipation of the aerosols. This finding suggests that exposure to certain forms of acid air pollution may have the potential to cause permanent lung damage.

4. Are the health effects of acid rain precursors purely a medical problem? Is there an exclusively medical solution to these health risks? In the absence of new government regulation of air pollution, will the problem be cured through medical technology?

I do not see any purely medical solution to the health risks which are posed by acid air pollution. It would be unreasonable to ask children to wear respirators during the summer months. There is no medical treatment of which I am aware which would prevent or alleviate the adverse effects of pulmonary function which are produced by exposure to acid air pollution. Therefore, the only rational approach to this problem is through prevention. In this instance, the essence of prevention consists of limiting the amounts of acid air pollutants which enter the atmosphere. I see no option to accomplish that end except government regulation.

5. Are there any studies which document the health effects of air pollution on neonatal health? Are you able to draw any conclusions about the effect of air pollution on neonatal health? If so, please describe.

I am aware of no studies which specifically document the health effects of air pollution on neonatal health. However, it is well known that the lungs of newborn infants are more vulnerable to many insults than the lungs of older children or adults. Therefore, it is prudent to suppose that neonates are particularly vulnerable to acid air pollution and ought to be protected accordingly.

Additional Questions from Senator Max Baucus

1. The testimony presented at this hearing encompassed a wide range of children, from the unborn fetus to teenagers of 17 years of age. Is any specific group affected more adversely than another?

Within the pediatric age group, it is well recognized that neonates and young children under the age of 4 are particularly vulnerable to respiratory insults. This heightened vulnerability results from the extremely narrow diameter of the airways in young children; these narrow airways can easily become blocked by any form of reactive inflammation stimulated by infection, toxic exposure, or other cause.

2. Please describe the medical picture of a child suffering from chronic bronchitis or asthma.

The classic medical picture of a child suffering from asthma or bronchitis is wheezing and difficulty in obtaining air. Typically, such children present to an emergeny room complaining of progressive chest tightness of several hours or days duration. On physical examination, wheezes are heard throughout the chest and movement of air is diminished. Frequently, such children are pale or even blue in color. Retraction of the skin above the collarbones and between the ribs is evident as they struggle to breathe. Acute asthma can be a life-threatening condition. Fortunately, asthma is a preventable condition. The frequency of asthmatic attacks can be reduced by reducing children's exposure to environmental factors, which trigger attacks, including air pollution.

3. Are we seeing any changes in asthma and bronchitis attacks throughout the country?

Data from numerous sources indicates that the frequency of attacks and hospital admissions from asthma and bronchitis is increasing throughout the country. The causes of this increase are not entirely clear. Air pollution would appear to be one among many factors responsible for the increase.

4. As noted in the written testimony, 21% of all hospitalizations of children under 15 years of age in the United States in 1980 were due to pulmonary disease. How do these figures vary for different states or regions of the country? How do they vary between urban and rural areas? What about a state like Montana?

I have no data on these questions.

5. Testimony also noted that 4.8 million days of-care, or 29% of total hospital days for children under the age of 15, is for pulmonary disease. What is the cost of this care?

I have no data on this question.

6. In your opinion, how would the lives of children suffering from respiratory distress be changed by reducing the ambient levels of acid rain precursors? How would this translate in terms of a reduction the number of hospitalizations for pediatric pulmonary disease?

   In my opinion, the lives of children suffering from respiratory distress would unquestionably be changed for the better by reducing ambient levels of acid air pollution. Acid air pollution has clearly been associated with increased frequency of asthma attacks and decreased respiratory function. An asthmatic attack is a dreadful event for a child involving as it does the struggle to breath, the threat of hospitalization and death, and the need for painful injections. Any regulatory measure which can reduce the frequency of childrens' admissions to hospital for this condition is for the good.

7. What type of long-term damage is being caused in children breathing air pollution?

   Sufficient long-term data are not yet available to determine whether the respiratory effects of acid air pollution involve permanent damage to the lungs. However, it is important to note that the adverse effects on pulmonary function which were observed to occur in children in summer camp following exposure to acid aerosols persisted for as long as four to five days after dissipation of the acid aerosols. This finding suggests that exposure to certain forms of acid air pollution may have the potential to produce permanent lung damage in children.

8. Does air pollution cause a child's immune system to be less effective in warding off disease, or does a less developed immune system allow for greater incidence of air pollution-induced infections?

   I have no information on this point.

9. Do we know enough about the public health problems caused by air pollution for the standards to be tightened?

   In my opinion, sufficient new information has arisen in the last five years on the health effects of acid air pollution to justify immediate tightening of current standards.

10. The Clean Air Act requires EPA to review air standards every three years. Is this review occurring? If it were, would EPA be compelled to tighten the standards?

    I have no information on whether EPA is periodically reviewing standards. In any event, however, EPA ought in my opinion be compelled to tighten current standards.

11. EPA is either unwilling or unable to set a standard that protects public health. Should Congress statutorily set this standard?

    Ideally, EPA ought to set a standard protective of public health under the provisions of the Clean Air Act. I should think that the first objective of the Congress should be to encourage EPA to obey the existing law and take such action. If, however, EPA is unwilling to do so, Congress should give strong consideration to setting a standard by statute. A clear precedent for Congressional standard setting exists in the coal dust standard for underground mines established by Congressional action under the Mine Safety and Health Act.

12. You indicated that there have been striking advance in our knowledge of the health effects of air pollution. Briefly, what are some of these findings? Has EPA reviewed these studies? Will you supply a detailed list of these studies to the Committee?

    Several lines of converging evidence - epidemiologic studies of populations, controlled studies of human volunteers, and toxicologic studies in cell systems and animals - all indicate that current levels of acid air pollution are able to produce substantial adverse health effects in certain segments of the American population, and particularly in children. I reviewed this research on pages 2 through 5 of my written testimony. The key reference which describes and summarizes this research is a report on a workshop on the Health Effects of Air Pollution which was convened by the National Institute of Environmental Health Sciences (NIEHS) in January 1987. This document is up-to-date, clearly written and extensively referenced. It contains the best compilation and critical assessment of the recent literature on this subject. I understand that EPA was represented at the NIEHS Workshop.

13. A large portion of the testimony has been devoted to the adverse health effects of acid deposition on children. Would you briefly describe the impact of acid deposition on the health of the elderly? What is the cost to Medicare?

    The elderly, like young children, constitute a subset of our population who are particularly vulnerable to the effects of acid air pollution. The elderly have less physiologic reserve than younger persons. Frequently the elderly have underlying respiratory disease as the result of previous infections, cigarette smoking or occupational exposures. For all of these reasons the elderly, particularly those with underlying respiratory disease, require special protection against acid air pollution.

    I have no data on the financial costs to Medicare of the exposure of the elderly to acid air pollution.

Additional Questions from Senator Frank R. Lautenberg.

1. Do you feel that current medical and scientific knowledge justifies Congress taking action now to control sulfates, nitrogen oxides, and ozone?

   Ample scientific evidence developed over the past five years indicates that current levels of exposure to acid air pollutants are sufficient to produce respiratory impairment in vulnerable subgroups within our population. These groups include young children, the elderly, and persons with cardiorespiratory disease. For that reason, I feel that sufficient evidence exists now to justify the Congress taking action to control sulfates, oxides of nitrogen and ozone.

2. You have commented on the health effects caused by the pollutants associated with acid rain. You have limited your comments, I assume, to the effects caused by current air quality levels. If left unchecked, do you expect the sulfate, NOx, and ozone levels to increase? If these levels increase, am I correct in assuming that the harms you have described would worsen? (i.e., would sensitive groups be more severely affected, and would more people suffer health effects?)

   I do not have any data as to whether current levels of sulfate, oxides of nitrogen and ozone are expected to increase; EPA will have that information. However, there is ample data from previous epidemiologic and laboratory studies to indicate that if levels of these pollutants do increase, the resultant adverse health effects will inevitably become more frequent and more severe. Clearly, then, steps must be taken to prevent any further increases in concentrations of these pollutants.

3. You have described health effects of the pollutants related to acid rain. I would like to get a concrete, layperson's sense of what kind of health effects we are discussing here. For example, will these pollutants actually cause or contribute to death in sensitive groups or in the population generally? Are any of the health effects you have described painful? Are the conditions or effects you have described permanent? Would these pollutants cause headaches, nausea, unconsciousness? Would the effects you have described affect someone's ability to do school work or perform a job? How many individuals in this country experience these harms or effects?

   The data which have been developed in the past several years indicate beyond any possibility of doubt that exposure to acid air pollutants causes increased frequency of respiratory disease in persons, particularly in the very young and the elderly, who are exposed to these pollutants. I am not aware of any data which indicate unequivocally that death has resulted from these exposures. However, it has been shown quite clearly that these exposures produce increased hospitalizations from respiratory disease and increased incidence of bronchitis and asthma.

Asthmatic attacks are painful and frightening events. They start by wheezing, by a struggle for breath, and by a feeling of imminent suffocation. They frequently require emergency hospitalization, sometimes of several days duration, and their treatment involves painful injections.

The currently available data do not indicate whether or not the adverse pulmonary effects produced by acid air pollution are permanent. However, the data do indicate that functional changes in the lung which are produced by exposure to acid aerosols, can persist for several days after dissipation of the acid aerosols. These data indicate therefore that acid air pollution has the potential to produce permanent lung damage.

Headaches and nausea could quite clearly result from exposure to acid air pollutants. Unconsciousness would not likely result, except possibly in the context of a severe asthmatic attack with reduction in the supply of oxygen to the brain.

The pulmonary effects produced by acid air pollution, such as asthma and bronchitis, could certainly impair a person's ability to do school work or perform a job.

I do not have precise data of the number of persons effected by these conditions. The population at risk is, however, very large.

4. Do you think the federal government is making a sufficient commitment to research on the causes and effects of acid rain? Could you describe the federal involvement? Is more federal funding required? Is the coordination of research efforts being done effectively by different federal agencies and departments?

In my opinion, the federal government is only now becoming aware of the research data which have been generated over the past five years pertaining to the health effects of acid air pollution. The agency which has most effectively reacted to date to this information is the National Institute of Environmental Health Sciences (NIEHS). Early in 1986, the NIEHS held a special symposium on the health effects of acid rain. This was a landmark event which brought together experts from around the nation to discuss the recently generated research findings. NIEHS has already produced preliminary proceedings from that meeting, and final proceedings are expected shortly. This expedited review of research findings by a federal agency is in the best traditions of the Public Health Service. It ought to be continued and extended. At the same time, the regulatory agencies, particularly the Environmental Protection Agency, must be encouraged to become closely acquainted with the recent research data and to take appropriate regulatory action without delay.

Thank you again. If you wish further clarification on any of these points, please do not hesitate to call upon me.

Sincerely,

Philip J. Landrigan, M.D.
Professor

PJL:ss

WRITTEN TESTIMONY OF CONSOLIDATION COAL COMPANY SUBMITTED FOR THE RECORD OF THE HEARING HELD FEBRUARY 3, 1987 BEFORE THE SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION

Consolidation Coal Company, a subsidiary of the Du Pont Company, is the second largest producer of coal in the United States. In 1986, Consol produced 41.5 million tons of coal in nine states and the province of Alberta. Consol opposes the enactment of acid rain legislation.

We are submitting this testimony for the record because we are concerned that the Subcommittee hearing on the health effects of air pollution held on February 3, 1987 did not provide a variety of views adequate to reach any dispassionate conclusion on this important issue. The four witnesses held a common point of view. However, their sweeping conclusions on a number of issues do not represent the views of other competent researchers in the field.

Based on the testimony received thus far, the Subcommittee will be unable to determine the relative contribution of any single air pollutant to the overall issue of health risks from air pollution. Nevertheless, such a determination should be made if the Committee intends to pursue legislative strategies which primarily focus on a single pollutant, sulfur dioxide. (This appears to be the case judging from the bills currently before the subcommittee on the subject.)

The witnesses for the February 3 hearing left the committee with the impression that large segments of the general population were being affected by air pollution. Whether or not that statement is true for the panoply of air pollutants (ozone, carbon monoxide, nitrogen oxides, sulfates, sulfur dioxide and others) taken collectively, our review of available literature on the subject suggests that, at least with regard to sulfur dioxide and sulfates, the general public health is not being adversely affected at current ambient levels. This statement is true for short term, long term and peak exposures.

We believe the evidence overwhelmingly shows that normally healthy persons are more than adequately protected by the Clean Air Act's current primary standard for sulfur dioxide. However, the Subcommittee reasonably may inquire about the effects on individuals with respiratory impairments. A review of the available literature shows that most researchers have concluded that asthmatics are the group within the general population most likely to experience health-related effects from exposure to air pollution.

With regard to sulfur dioxide, human clinical studies suggest that SO_2-induced responses would occur in asthmatics who are exposed to peak SO_2 levels (above 0.4-0.5 ppm) during moderate or heavy exercise AND who are sufficiently sensitive to SO_2 to experience a response AND who are not taking medication that would prevent a response. Under current Clean Air Act standards, however, such ambient levels of SO_2 rarely occur.

Exposures to peak SO2 concentrations of 0.4-0.5 ppm or higher for periods of ten minutes or more are likely to occur only in the vicinity of major point sources of SO_2 and only outdoors. Most asthmatics would have very little chance for such exposures. Moreover, even for asthmatics in the vicinity of such sources (estimated to be 25% of asthmatics), the effects of such exposure occur only during exercise -- outdoors -- at a moderate to high rate.

The remainder of this testimony reviews in greater detail the available information regarding the effects of SO_2 on public health. Much of this testimony focuses on the effects of SO_2 on asthmatics because the available evidence demonstrates that normally healthy persons do not experience effects on respiratory or pulmonary function from current ambient levels of SO_2. To reiterate, the present Clean Air Act primary standard for SO_2 is protecting public health.

EVIDENCE RELATING TO THE EFFECTS OF LONG-TERM EXPOSURES TO SULFUR OXIDES

Statistical Studies of Mortality Rates

During the past 15 years, several investigators have conducted large-scale statistical studies using multiple regression analysis to evaluate possible associations between annual mortality rates in large numbers of metropolitan areas in the U.S. and various indices of air pollution in those areas. Some of those studies have found statistical associations between increased mortality and certain pollutants, including sulfates and other particles (e.g., Lave and Seskin 1977, Chappie and Lave 1982, Mendelsohn and Orcutt 1979, Ozkaynak and Spengler 1985); while others have failed to confirm those results (e.g., Lipfert 1984, Selvin et al. 1984). Based on an interpretation of these studies, some have claimed that long-term exposure to coal-related air pollutants, including sulfates, can cause increased mortality. For example, a 1984 report by the Congressional Office of Technology Assessment asserted, based on such an interpretation, that the possible health risk posed by current levels of sulfates and other particulates could be estimated at 50,000 premature deaths per year in the United States and Canada (OTA 1984).

These studies, however, have been widely criticized by the scientific community, particularly for their failure to take adequate account of potentially confounding variables that could have influenced the results. Hence, they are not considered reliable or useful for assessing the health effects of sulfates.

EPA has noted many of the limitations of these studies, as well as many key unresolved issues concerning the reported associations and whether they are causal or not, and has declined to put any reliance on these studies (EPA 1986a). Numerous reviewers have likewise pointed out that these studies do not permit reliable conclusions as to the mortality effects of long-term exposure to sulfates or other air pollutants, and certainly do not provide any valid basis whatsoever for the kind of estimates made by the OTA report (e.g., Lipfert 1986a, Higgins 1986, Ware et al. 1979). For these reasons this group of studies cannot be used for standard-setting or policy-making purposes.

Studies on Chronic Morbidity Effects

Data from England in the 1960's showed an increased frequency of respiratory symptoms, as well as decreased lung function, in children exposed to high annual-average levels of SO_2 (181-275 $\mu g/m^3$) accompanied by high levels of particulate matter (230-301 $\mu g/m^3$ of British Smoke) (Lunn et al. 1967). Several more recent studies from the U.S. have reported an increased prevalence of cough and other respiratory symptoms -- but not lung function effects -- associated with long-term exposure to SO_2 and particulate matter in urban areas (Bouhuys et al. 1978, Ware et al. 1986) or to SO_2 alone in areas around large point sources of SO_2 (Chapman et al. 1985, Dodge 1983, Dodge et al. 1985, Schenker et al. 1983). These studies, however, do not provide any objective evidence of an effect of SO_2 at current ambient levels on respiratory health.

Both Bouhuys et al. (1978) and Ware et al. (1986) found a mixture of positive and negative results. As noted both by the authors themselves and by several reviewers (e.g. Lipfert 1986a, Higgins 1986, Roth 1986, Holland et al. 1986), these inconsistencies, as well as the investigators' failure to take account of potentially confounding factors, make it impossible to attribute the increased symptoms to either SO_2 or particulate matter. Similarly, the reviewers have explained that, due to several problems with the studies conducted around large SO_2 sources, those studies do not establish whether the apparent perception of increased respiratory symptoms in such areas is in fact real or related to SO_2 exposures (see e.g., Lipfert 1986a, Higgins 1986, Roth 1986). In any event, the annual-average SO_2 concentrations implicated in the latter studies were above the level of the current annual-average standard. In short, the recent U.S. studies have found no effect of long-term SO_2 exposure on lung function, and the reported associations of pollution with symptoms do not provide reliable evidence of an effect of SO_2, at least at concentrations below the current annual standard.

A couple of recent European studies have reported an increased prevalence of symptoms and lung function effects associated with long-term SO_2 exposures (PAARC 1982a & b, van der Lende et al. 1986). However, several uncertainties, inconsistencies, and unanswered questions about these studies preclude acceptance of those reported findings (see EPA 1986a & 1986b, Lipfert 1986a, Higgins 1986, Holland et al. 1986a, Roth 1986).

Conclusions : Current Standards Adequate

Based on the studies of effects associated with long-term exposures to sulfur oxides, it appears that if an annual-average SO_2 standard is needed at all, the current standard is entirely adequate to protect the public health. Moreover, there is no reliable evidence that long-term exposure to sulfates at ambient levels causes adverse health effects.

EVIDENCE RELATING TO EFFECTS OF 24-HOUR EXPOSURES TO SULFUR OXIDES

Mortality Data

There is a large body of data on daily mortality rates and daily pollution levels in London during the winters of 1958/59 through 1971/72. These data have been analyzed frequently by numerous statistical experts (e.g., Ware et al. 1981, Mazumdar et al. 1981 & 1982, Shumway et al. 1983, Ostro 1984, Schwartz and Marcus 1986). It is generally agreed that these analyses indicate an increased risk of mortality associated with 24-hour concentrations of both SO_2 and particulate matter (measured as British Smoke) in the range of 500 to 1000 $\mu g/m^3$, most clearly at levels in excess of about 700-750 $\mu g/m^3$, but that the effects of SO_2 and particulate matter cannot be distinguished from each other or from other possible causal factors (EPA 1986a & 1986b, Holland et al. 1986a & b, Lawther 1985, Higgins 1986, Roth 1986). Although there is dispute as to whether the analyses also suggest an increased risk of mortality at 24-hour British Smoke levels below 500 $\mu g/m^3$,1/ there is general agreement that they do not suggest such effects at SO_2 levels below 500 $\mu g/m^3$. Indeed, the analyses by Mazumdar et al. (1982) and by Schwartz and Marcus (1986) found that SO_2 is not an important explanatory variable in assessing the increased daily mortality in London during the winters in question, at least at concentrations below about 700 $\mu g/m^3$.

Moreover, air quality conditions in London during the winters in question were quite different from those currently found in the United States in terms of pollution composition (e.g., fog), meteorological patterns, indoor sources, and other factors. Hence, the London results cannot be extrapolated to predict effects at comparable concentrations in the U. S. today.

Other recent studies examined daily pollutant-mortality relationships in more contemporary atmospheres in New York City (Ozkaynak and Spengler 1985, Ozkaynak et al. 1986), Pittsburgh (Mazumdar and Sussman 1983), and Athens, Greece (Hatzakis et al. 1986). Mazumdar and Sussman (1983) found no effect on SO_2 on mortality in Pittsburgh, and the studies in New York and Athens involve too many problems and unresolved questions to provide any reliable evidence of an association between SO_2 and mortality (see EPA 1986a & 1986b, Lipfert 1986a, Higgins 1986, Holland et al. 1986a).

Morbidity Studies

It is widely accepted that the most reliable data on the morbidity effects of daily SO_2 and particulate matter levels derive from the studies of Lawther et al. (1970) of exacerbations in the health status of chronic bronchitis patients in London in the 1950's and 1960's. These studies showed such effects at daily concentrations at and above 500-600 $\mu g/m^3$ SO_2 and 250-300 $\mu g/m^3$ British Smoke.

More recent studies by Dockery et al. (1982) and Dassen et al. (1986) have investigated the pulmonary function of schoolchildren in Steubenville, Ohio, and the Netherlands, respectively, in relation to daily SO_2 and particulate levels before, during, and after air pollution episodes. These studies reported small, reversible declines in the children's lung function following episodes in which the 24-hour concentrations reached 250-450 $\mu g/m^3$ of SO_2 and 200-420 $\mu g/m^3$ of particulate matter. These studies, however, do not provide a sound scientific basis for setting air quality standards. The mean lung function declines reported were within the range of an individual's "natural" daily and seasonal variations in lung function (see Lipfert 1986b). Many medical experts believe that such small, reversible changes in lung function are not adverse health effects. Moreover, there are substantial uncertainties as to whether the effects reported in these studies were in fact related to SO_2 or particulate matter exposures at all. These uncertainties arise from a number of inconsistencies in the findings, unresolved questions about the results, possible confounding factors that were not adequately taken into account, and problems in the data analysis (see e.g., Lipfert 1986b, Holland et al. 1986b, Higgins 1986, Roth 1986). Due to such uncertainties, these studies do not provide reliable evidence of an effect of pollution on health.

A few other recent studies have purported to implicate sulfates or fine particles generally as related to morbidity effects. For example, Bates and Sixto (1983, 1986) reported an association in summer (but not winter) between hospital admissions in Southern Ontario and daily sulfate and ozone levels, while Ostro (in press 1986) reported an association of work loss and restricted activity in adults in various U.S. cities with fine particle levels 2-4 weeks earlier. As pointed out by EPA and several reviewers, however, these studies involve numerous problems and uncertainties, which preclude attribution of the reported effects to the pollutants studied and thus preclude reliance on the studies for standard-setting or policy-making purposes (EPA 1986a, Lipfert 1986a, Holland et al. 1986a, Higgins 1986, Chai 1986).

Conclusions : Current 24 Hour Standard Wholly Adequate

The studies of the acute effects of daily pollution exposures do not provide any reliable evidence that exposure to 24-hour SO_2 concentrations below 500 ug/m^3 are related to adverse health effects. Accordingly, it is clear that the current 24-hour SO_2 standard of 365 ug/m^3 protects the public health from such acute effects with a wholly adequate, if not more than adequate, margin of safety. In addition, there has been no credible showing that exposure to sulfates at current ambient levels is related to any acute health effects.

EVIDENCE RELATING TO EFFECTS OF PEAK EXPOSURES TO SULFUR OXIDES

Findings from Human Clinical Studies

The evidence on the effects of brief peak exposures to sulfur oxides, including SO_2 and sulfuric acid aerosol, comes from human clinical studies using relatively few subjects, rather than from epidemiological or other real-world evidence on actual health effects in the community environment. The discussion here focuses mainly on the evidence from clinical studies of SO_2 effects; however, mention is also made of studies on the effects of sulfuric acid aerosol.

Effects of SO_2

A number of human clinical studies from several laboratories in the past 5-6 years have investigated the effects of SO_2 on respiratory function. The findings from these studies are summarized in Chapter 4 of EPA's Criteria Document Addendum (EPA 1986a) and on pp. 9-19 of the draft Staff Paper Addendum (EPA 1986b), and are discussed by McFadden (1986a & 1986b), Ingram (1986), Chai (1986), and Proctor (1985). In general, these studies show the following:

(a) Normal healthy subjects do not experience effects on respiratory or pulmonary function from short-term exposures to SO_2 concentrations up to about 2.0 ppm. Effects have been seen at these levels only in asthmatics.

(b) At SO_2 concentrations of 1.0 ppm and below, simply exposing asthmatics to SO_2 has not produced effects. Rather, in order for effects to occur in asthmatics at these concentrations, the SO_2 exposure must be accompanied by moderate to heavy exercise or some other form of increased ventilation by mouth, which increases the effective "dose" of SO_2 to the respiratory tract. This complicates the interpretation, since either exercise or mouth breathing alone can produce bronchoconstriction in asthmatics.

(c) Exposure of exercising asthmatics to SO_2 concentrations in the range of about 0.4-0,5 ppm to about 1.0 ppm has produced statistically significant responses indicative of bronchoconstriction in the asthmatics.2/ These responses consist of transient changes in certain sensitive tests of pulmonary function, such as specific airway resistance ("SRaw"), and in some cases transitory symptomatic effects, such as wheezing, shortness of breath, or chest tightness. These responses are reversible and short-lived. They appear within the first 5-10 minutes of the onset of the exposure and exercise, do not get significantly worse with continued exposure or exercise, and disappear promptly following cessation of the exposure or exercise. As several studies have found, recovery from these effects generally occurs well within one hour after the cessation of exercise, even when the SO_2 exposure is continuing (Hackney *et al*. 1984, Linn *et al*. 1984c, Schachter *et al*. 1984). Consecutive exposures repeated within less than one hour after the original exposure produce a lesser response (Sheppard *et al*. 1983, Roger *et al*. 1985, Kehrl *et al*. 1986), but exposures repeated 5-24 hours later show no different effect than the original exposure (Sheppard *et al*. 1983, Linn *et al*. 1984c). These findings indicate that SO_2 exposures do not sensitize the airways to this pollutant or change airway reactivity (see McFadden 1986a & 1986b). Other studies have similarly found no residual effect of SO_2 exposure in the day or week after the exposure (Linn *et al*. 1983b, Hackney *et al*. 1984, Witek *et al*. 1985).

(d) In general, the group responses observed in these studies show a dose-response relationship with the level of SO_2. There is, however, substantial variability in asthmatics' responses to SO_2 at these concentrations, with some asthmatic subjects not responding at all. For example, an analysis by Horstman *et al*. (1986a), using an increase of 100% or more in SRaw as a benchmark, found that only about 25% of the exercising asthmatics studied showed such a response at 0.5 ppm and that only about half of them showed such a response even at 1.0 ppm. The findings of Schachter *et al*. (1984) show similar results for a 10-15% decrease in forced expiratory volume in one second ("FEV_1"), a common lung function test. Likewise, in nine studies in which the authors reported instances in which particular SO_2-exposed asthmatics used a bronchodilator after the exposure to relieve symptoms or had to cease the exposure protocol or had an unusually large or prolonged (*e.g.*, greater than 45 minutes) functional response, such reactions were reported to have occurred, in the aggregate, in only 15 out of 150 subjects. See Linn *et al*. (1983b, 1984a, 1984b, 1984c), Bethel *et al*. (1984), Roger *et al*. (1985), Koenig *et al*. (1985), Sheppard *et al*. (1983), and Schachter *et al*. (1984).

(e) The studies to date have generally utilized mild asthmatics as subjects. There is no evidence as to the potential effects of SO_2 exposure on more severe asthmatics. One study, however, did investigate the effects of SO_2 on patients with chronic obstructive pulmonary disease ("COPD"), and found no effects of SO_2 concentrations of 0.4 and 0.8 ppm on such patients who were exercising lightly (Linn et al. 1985b). This finding is important both because COPD patients appear from the epidemiological studies (e.g., Lawther et al. 1970) to be the persons at greatest risk of serious health effects from high SO_2/particulate levels, and because the finding of no effect in these patients may also be applicable to severely impaired asthmatics.3/

Effects of sulfuric acid aerosol and other sulfates

In general, the human clinical studies on the effects of sulfuric acid aerosol ("H_2SO_4") have produced results that are qualitatively similar to those for SO_2 in certain respects. At H_2SO_4 concentrations below about 1000 ug/m^3, statistically significant pulmonary effects have not been found in normal subjects (Utell et al. 1984 & 1986, Horvath et al. 1982, Horstman et al. 1982, Avol et al. 1979), but only in asthmatics who were exercising or breathing through a mouthpiece. Utell et al. 1983, 1984, 1986) observed such responses in exercising or mouth-breathing asthmatics at an H_2SO_4 concentration of 450 ug/m^3, while Linn et al. (1986) did not find any statistically significant effects in exercising asthmatics at about this level. At lower levels of H_2SO_4, effects have generally not been found (Utell et al. 1983, 1984 & 1986, Linn et al. 1986, Horstman et al. 1986b, Spektor et al. 1985, Avol et al. 1979), although a single study reported small responses in exercising asthmatics exposed to 100 ug/m^3 via mouthpiece (Koenig et al. 1983). The responses that have been observed in these studies consist of small, reversible, and short-lived changes in pulmonary function. These studies have not reported increased respiratory symptoms attributable to H_2SO_4 exposure.

The studies also indicate that other sulfates (e.g., ammonium sulfate, ammonium bisulfate) are less irritant than H_2SO_4. Thus, exposure to such other sulfates at levels below 1000 ug/m^3 has not produced increased responses even in mouth-breathing asthmatics (Utell et al. 1983 & 1984).

It should be noted that, in the ambient air, concentrations of total sulfates rarely, if ever, approach even 100 ug/m^3, and that of the total sulfates, only a small fraction consists of the acid sulfate species, with H_2SO_4 composing only about 3-5% of the total (Kittleman et al. 1983).

Health Significance of Responses in Exercising Asthmatics

The findings of statistically significant responses in exercising asthmatics exposed to SO_2 (or H_2SO_4) do not mean that such responses are of significance to the asthmatics' health. EPA's draft Staff Paper Addendum explicitly recognizes that the health significance of the reported responses to SO_2 is "open to question," and that the scientific literature does not provide sufficient information to specify an SO_2 concentration at which the observed effects can be deemed adverse or indicative of adverse

health effects. There are, however, a number of factors that should be considered in evaluating the health significance of the reported or anticipated responses of exercising asthmatics.

(a) The changes in pulmonary function observed in the studies are not in themselves significant to the asthmatics' health. Those changes are modest, transient, and quickly reversible. As explained by Drs. McFadden (1986a & 1986b) and Chai (1986), both of whom are experts on asthma, changes of 100-200% in SRaw and changes of up to 15-20% in FEV_1 -- which are the kinds of changes typically observed in these studies -- are common in asthmatics, are within the range of lung function variations experienced by asthmatics in response to a wide variety of frequently encountered stimuli, and do not represent a significant health effect. By way of comparison, as noted by McFadden (1986b), asthmatic patients coming to an emergency room for treatment frequently show increases of 900-1000% in SRaw.

(b) As a general matter, the reported symptomic effects (e.g., coughing, shortness of breath, chest tightness) are temporary and short-lived, abating soon after the cessation of exercise. They are comparable to or less than the reactions of asthmatics to a wide variety of stimuli and conditions (e.g., exercise or cold air alone, laughing or sighing, exposure to commonly found irritants, etc.) which asthmatics frequently encounter and generally tolerate well; they do not lead to acute attacks requiring emergency therapy hospitalization for relief; and they do not appear to have any cumulative or long-term effects (see McFadden 1986a & 1986b, Chai 1986, Ingram 1986, Proctor 1985). Thus, for asthmatics generally, any symptomatic responses to peak SO_2 exposures during moderate or higher exercise would be, at most, temporary annoyances.

(c) As noted above, some of the studies have reported that a few individual asthmatics exposed to SO_2 showed larger or longer-lasting changes than the rest or experienced reactions calling for treatment with a bronchodilator or removal from the exposure protocol. Even these reports, however, do not demonstrate serious health effects. For example, use of an inhaled bronchodilator is an ordinary part of the asthmatics' experience and cannot be equated with the kind of medical treatment that would indicate a significant health effect (McFadden 1986a). Even the very few subjects who had to discontinue the exposures did not have to be hospitalized or to take unusual medication; their bronchoconstriction reversed promptly when they ceased the exposure and inhaled a routine bronchodilator. Moreover, it is remarkable how few such reactions have been reported in studies in which asthmatics (some of whom were pre-selected for sensitivity to SO_2) were exposed during moderate to heavy exercise to peak SO_2 levels that were expected to produce a bronchoconstrictive response. As pointed out above, the great majority of asthmatics so exposed (90% in nine studies in which some such reactions were reported) did not show such effects.

(d) There is no evidence whatsoever that these short-term responses have any cumulative or long-term health consequences. Indeed, there is considerable evidence that they do not. For example, the studies finding no greater responses to SO_2 when subjects were reexposed on subsequent occasions and those finding no residual effect of the SO_2 exposures in the day or week after the exposures indicate that SO_2 does not alter airway reactivity in a way that would lead to recurrent episodes of bronchoconstriction over a prolonged period and does not have any other long-term effect. Moreover, there is no indication from either clinical or epidemiological studies that short-term SO_2 exposures exacerbate the asthmatics' underlying condition or lead to any other chronic harm to asthmatics' health status.

(e) In evaluating the health significance of the reported responses of exercising asthmatics to peak SO_2 exposures, it should be noted that a large number of medical experts, including several well-known experts on asthma, have expressed the opinion that those responses are not themselves, and are not indicative of, adverse health effects. These experts include Drs. McFadden (1986a & 1986b), Ingram (1986), Chai (1986), Proctor (1985), Higgins (1986), and Lawther (1982a & 1982b).

(f) EPA's draft Staff Paper Addendum points out that most studies have used mild asthmatics, and it expresses concern that, despite the range of SO_2-sensitivity shown in the studies, even more sensitive individuals may exist in the population of mild asthmatics and that more severe asthmatics may be more sensitive to SO_2-induced bronchoconstriction (EPA 1986b, p. 35). These are speculations, however; there are no data whatever to support either of these concerns. Indeed, as several experts have pointed out, there is good reason to believe that severely impaired asthmatics would not be more sensitive to, or suffer more serious effects from, peak SO_2 exposures, because of the limitations on their ability to achieve sufficiently high exercise rates without medication (McFadden 1986a & 1986b, Ingram 1986, Chai 1986, Proctor 1985, Higgins 1986). In particular, it is unlikely that asthmatics with severely impaired lung function would be able to achieve the relatively high ventilation through exercise that appears to be necessary, in conjunction with SO_2 exposure, to produce a response. And even if they could, they would most likely be able to do so only through the use of a bronchodilator or other medication, which would totally prevent or eliminate any SO_2-induced response. Thus, severely impaired asthmatics may, paradoxically, be less responsive to SO_2 than the mild asthmatics used in the studies.

Lack of epidemiological evidence

Epidemiological evidence is considered to be "real-world" evidence as contrasted with clinical studies which are conducted in artificially control laboratory settings. There is no reliable epidemiological evidence of an association between SO_2 (or sulfates) at current ambient levels and increased frequency or severity of asthmatic attacks or other adverse health consequences in asthmatics in the general population (Chai 1986, Higgins 1986, Ingram 1986, McFadden 1986b, Lawther 1982b, Proctor 1985). Indeed, some studies have found no such relationship. In an important study recently published, Goldstein and Weinstein (1986) found no association between short-term SO_2 peaks above various thresholds (0.5, 0.3, and 0.1 ppm) in certain areas of New York City in the period 1969-1971 and emergency room visits for asthma in those areas. Other studies also have found no relationship between SO_2 and health effects in asthmatics (Goldstein and Dulberg 1981, Dodge 1983, Dodge et al. 1985).

Before the responses reported in asthmatics in the laboratory studies are accepted as showing or being indicative of effects of health significance, there should at least be some reliable evidence of an actual public health impact, in the form of real-world data showing the occurrence of adverse health effects in asthmatics due to exposures to SO_2 in the ambient air. To date, no such evidence exists.

Exposure probabilities

Based on the human clinical studies, it would appear that SO_2 -induced responses would occur only in asthmatics who are exposed to peak SO_2 levels above about 0.4-0.5 ppm during moderate or heavy exercise, who are sufficiently sensitive to SO_2 to experience a response, and who are not taking medication that would prevent a response. The available data indicate that, under the existing set of SO_2 standards, such situations would occur very rarely in the ambient environment.

With current air quality, exposures to peak SO_2 concentrations of 0.4-0.5 ppm or higher for periods of 5-10 minutes or longer are likely to occur only in the vicinity of major point sources of SO_2 and only outdoors (EPA 1986b, pp. 37-38). Thus, the only asthmatics potentially subject to such exposures are those in the vicinity of major sources that could produce such peaks. These represent only a fraction of all asthmatics in the United States (about 25%), which means that for the majority of asthmatics in the country the chance of such exposure is virtually nil.

Moreover, even for asthmatics in the vicinity of such sources, the exposures potentially leading to a response would occur only for asthmatics who are exercising outdoors at a sufficient rate to produce a response and who come into contact with a 5-10 minute peak of SO_2 at a sufficiently high level to produce a response in conjunction with the exercise. EPA has carried out an exposure analysis which estimated the probabilities that asthmatics living in the vicinity of four representative power plants would experience a five-minute exposure to 0.5 ppm SO_2 while at moderate or higher exercise (EPA 1986c). That analysis found that the percentage of such asthmatics estimated to experience at least one such exposure per year ranged from 0.2% (at the "lowest exposure" plant) to 13% (at the "worst case" plant studied) with current emissions, and from 2.9 to 14% if the existing standards were just met at these plants. The analysis found further that the percentages of such asthmatics estimated to have more than one such exposure per year would be substantially lower -- 0-4.8% calculated to have two or more such exposures and 0-2% calculated to have three or more such exposures. The analysis also noted that all these results likely overestimate the actual probabilities of exposure in several respects.4/

While the results from EPA's exposure analysis cannot be directly extrapolated to the population around other major sources, one can nevertheless use these figures as rough boundaries for the fraction of asthmatics around all such sources that would encounter such an exposure. It seems reasonable to assume further that approximately 25% of all asthmatics in the U.S. live in the vicinity of such sources that could produce SO_2 peaks at the levels of interest here.5/ Using these assumptions, the figures from the exposure analysis would indicate that the fraction of all asthmatics that would encounter such an exposure during exercise under the current standards would be between 0.05% (0.2% x 25%) and 3.5% (14% x 25%). Due to the overestimates in EPA's exposure analysis, the actual figure would probably be closer to the lower bound of this range than to the upper bound.

Moreover, not all asthmatics exposed during exercise to the SO_2 concentrations in question will experience a response. Only a portion of them will. For example, as mentioned above, the analysis by Horstman et al. (1986a) indicates that only about 25% of exercising asthmatics would experience a doubling in SRaw at 0.5 ppm SO_2. Using this finding, one could calculate that the fraction of all asthmatics who would have such a response at 0.5 ppm would lie between 0.012 and 0.88%. As also discussed above, however, a doubling in SRaw is not in itself a significant health effect. Using the incidence of reports, from the clinical studies, of bronchodilator usage, removal from the exposure protocol, or an unusually large or prolonged functional response -- about 10% -- would lead to an estimate of a fraction between 0.005 and 0.35% for such effects in all asthmatics. While these percentages are not precise estimates, they do illustrate the very small fraction of asthmatics who could experience such effects, even once per year, due to exposure to peak levels of SO_2 in the real world.6/

Finally, it should be noted that, while no similar exposure analysis has been conducted to estimate the probabilities of exposures or responses of exercising asthmatics to peak concentrations of sulfates, the probabilities would almost certainly be lower than those for SO_2, since the peak sulfate concentration levels used in the clinical studies are seldom, if ever, reached in the ambient air.

Relative effect of SO_2 exposure compared to other stimuli

In contrast to the very low probability and estimated incidence of responses in exercising asthmatics due to peak SO_2 (or sulfate) exposures, asthmatics commonly and frequently encounter other stimuli that can produce similar or greater responses. Various asthma experts have pointed out that asthmatics can have similar or stronger bronchoconstrictive reactions to a great variety of much more frequently encountered stimuli, including exercise alone, cold air alone, laughing, sighing, coughing, and exposure to numerous other agents such as naturally occurring allergens, perfume, powdered laundry detergents, other irritant pollutants, etc. (McFadden 1986a & 1986b, Ingram 1986, Chai 1986, Proctor 1985). It is important to assess the possible SO_2-induced effects in relation to and in the context of those other stimuli, in order to place such effects in perspective and to evaluate the overall significance of such SO_2-induced effects to asthmatics' health and quality of life.

For example, because asthmatics frequently and regularly encounter such other stimuli in their daily lives and experience a bronchoconstrictive response to at lease some of them, they learn to tolerate and cope with such responses -- either with or without medication. EPA's exposure analysis indicates that, with current air quality, the majority of asthmatics will never experience an SO_2 exposure sufficient to produce a response, and the relatively few that do so will encounter such an exposure during moderate or greater exercise only once or, at most, a few times a year. In the overall context of the many influences producing bronchoconstriction in asthmatics, the possibility of one or two peak SO_2 exposures per year, even assuming that they induce a response, would not have an appreciable incremental impact on the asthmatics' health, lifestyle, or quality of life. Thus, the possibility of such peak exposures would not appear to present a public health problem.

CONCLUSION

Evidence suggests that the only population group that responds to brief peak exposures are asthmatics, and then only during moderate to heavy exercise. Because not all asthmatics live near major sources of SO_2, EPA exposure analysis shows that between 0.2% and 14% of asthmatics could experience a 5-10 minute exposure to 0.5 ppm SO_2 during moderate to heavy exercise once a year. The responses are generally mild, short-lived and reversible. They do not require medication (other than, in a few cases, routine use of a bronchodilator) and do not lead to asthmatic attacks that require emergency therapy or hospitalization. Finally, they do not have or lead to any cummulative or long-term effects.

COUNTERVAILING HEALTH CONSIDERATIONS

In considering the public health risks raised by the evidence pertaining to sulfur dioxide, Congress cannot ignore the fact that adoption of stringent new standards could itself entail unnecessary risks to the public health. Implementation of SO_2 emissions reduction legislation would substantially increase the SO_2 control costs for coal-fired electrical utilities, and such increases in turn could lead to increases in the costs of home heating and cooling. These cost increases could result in more poorly heated homes in winter and less use of air conditioning in summer, which can have very real and serious adverse health effects, perticularly for the aged, the poor, the chronically ill, and young children. In addition, such legislation could lead to loss of jobs in affected industries, such as the high-sulfur coal industry (and mining-related industries) and the domestic nonferrous metal smelting industry; and the unemployment rate has a consistent and significant effect on physical and mental health (Brenner 1976). While these health risks are not well quantified, they further caution against setting new and stricter SO_2 emissions standards where the existing ambient standards for sulfur dioxide are adequately protecting public health.

SUMMARY

We have attempted to review the available literature and to discuss the results of these studies with qualified researchers and practitioners in the field in order to gain a better understanding of the issue. We are attaching to this testimony the names and addresses of several researchers or practitioners upon whose work much of our discussion is based. We believe these investigators hold views markedly different from those of the witnesses testifying at the February 3, 1987 hearing. In the interest of building a balanced hearing record on this subject, we encourage the Subcommittee to hold additional hearings to seek these differing views.

FOOTNOTES

1/ For example, EPA has stated that these analyses of the London mortality data suggest possible slight mortality risks at British Smoke ("BS") levels below 500 ug/m^3 (EPA 1986a & 1986b). However, numerous health effects experts, including the original investigators, have pointed out that these analyses do *not* indicate a causal exposure-effect relationship between BS and mortality at these lower BS levels (*e.g.*, Holland *et al*. 1986a & b, Lawther 1985, Lipfert 1986b, Higgins 1986, Roth 1986).

2/ There is no reliable evidence of SO_2-induced responses in exercising asthmatics at concentrations below 0.4 ppm. Five studies have found no significant effects at these levels (Linn *et al*. 1983b, 1984a & 1984b, Schachter *et al*. 1984, Roger *et al*. 1985). While Bethel *et al*. (1985) reported an excess SO_2 effect at 0.25 ppm with heavy exercise, that effect is not significant under appropriate statistical tests, and the validity of that finding is further undercut by the investigators' finding of no excess effect of 0.25 ppm SO_2 at an even higher exercise level (see EPA 1986a, McFadden 1986a).

3/ Linn *et al*. (1985b) suggested that their finding of no effect may have been related to the fact that the exercise rate was kept low due to the patients' inability to achieve a higher rate. As they noted further, however, the same exercise limitations would apply to COPD patients in the general population. Moreover, these limitations would also apply to severely impaired asthmatics in the general population, since it is unlikely that such asthmatics could achieve a moderate to high exercise level without taking medication (which would eliminate any SO_2-induced effect). This suggests that such individuals with compromised lung function would not be affected by brief SO_2 exposures.

4/ One of the most significant sources of overestimation is the assumption in the exposure analysis that activity patterns and ventilation rates for asthmatics are the same as for the general population. This assumption undoubtedly overstates activity levels, and thus the exposures of concern, at least for more severe asthmatics (EPA 1986c, p. 2-18). Other sources of overestimation include the unrealistic assumption that the power plants analyzed were continuously operating at 100% capacity; error in the dispersion modeling (which would tend to overpredict ambient SO_2 concentrations; and the use of one-hour blocks of predicted constant exercise (which is in fact unlikely) (*id*., pp. 2-14, 2-15, 2-19). While the exposure analysis also notes a couple of sources of possible underestimation (*id*., pp. 2-15, 2-17), such underestimates would not appear to be substantial and would certainly not offset the overestimates indicated by the above-mentioned factors. Hence, there is a net overestimation of actual exposure probabilities. In addition, it should be noted that the power plants studied were located in or near population centers, whereas many power plants and other major sources are located in sparsely populated rural areas, where substantially fewer people will be exposed.

5/ It is our understanding that EPA has estimates indicating that the number of asthmatics living in the vicinity of coal-fired power plants (which are the principal sources of 5-10 minute SO_2 peaks of 0.5 ppm or higher and are the types of plants studied in the exposure analysis) could be as high as 2.27 million (Burton 1986). Since asthmatics are assumed to constitute about 4% of the U.S. population of 237 million -- or about 9,5 million -- this figure would represent nearly 24% of all asthmatics. While some other major sources may also produce such 5-10 minute SO_2 peaks, they would appear to be relatively few. Thus, even taking into account potential exposures around such sources, it seems unlikely that the fraction of asthmatics who would be in areas potentially affected by 5-10 minute peaks of 0.5 ppm or more would exceed about one-quarter of all asthmatics in the country.

6/ Additional work is needed to estimate with greater precision the nationwide percentage of asthmatics who would experience a response due to peak SO_2 exposure. The Utility Air Regulatory Group is sponsoring an analysis that could lead to a better estimate of this figure. In the meantime, a rough boundary analysis such as that presented in the text shows the extremely small fraction of asthmatics who could experience effects from peak SO_2 exposures.

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